Dissertation on

Prospective Randomised Control Study of Dexmedetomidine for Controlled Hypotension in Functional Endoscopic Sinus Surgery (FESS)

Dissertation Submitted in partial fulfillment of

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BRANCH X – ANESTHESIOLOGY

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Certificate

This is to certify that this dissertation entitled **"Prospective Randomised Control Study of Dexmedetomidine for Controlled Hypotension in Functional Endoscopic Sinus Surgery (FESS)"** submitted by **Dr.R.Sugantha**, in partial fulfillment for the award of the degree of Doctor of Medicine in Anesthesiology by the Tamilnadu Dr.M.G.R. Medical University, Chennai is a bonafide record of the work done by her in the Department of Anesthesiology, Madras Medical College, during the academic year 2009 -2012.

Dr.V.Kanagasabai M.D.,
Dean,
Madras Medical College &
Govt. General Hospital,
Chennai-600 003

Prof. Dr.C.R.Kanyakumari,M.D,D.A,
Director,
Institute of Anesthesiology and
Critical care,
Madras Medical Collage,
Chennai – 600 003 .

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Contents

CHAPTER I

Introduction

Impairment of intra operative visibility due to bleeding is problem during otorhinolaryngologic surgeries especially in endoscopic surgeries like FESS. Bleeding in the surgical field can lead to incomplete surgical procedure which increases further bleeding and increased risk of complications due to non visualization of important structures. During these surgeries a slightest bleeding at the surgical area would look larger due to magnifying effect of the microscope which could upset surgical comfort. Controlled hypotension is one of the anesthetic techniques used to reduce bleeding during endoscopic surgeries. There are varieties of methods and medications used to obtained deliberate hypotension.

The ideal hypotensive agent should be non toxic, maintain cerebro vascular auto regulation, no change in cardiac function, have short term effect and be easily titrated[1, 2]. Alpha 2 agonists like clonidine augment hypotensive action and therefore reduce bleeding[3,4,5]. Dexmedetomidine another highly selective Alpha 2 agonist acts by central mechanism and reduces bleeding.

I have chosen this study to evaluate the effect of dexmedetomidine on the intraoperative isoflurane requirement to maintain mean arterial pressure of 60-70mmHg, quality of surgical field and awakening time in patients undergoing FESS.

CHAPTER II

Hypotensive anesthesia

Hypotensive anesthesia is a technique, used intra operatively to help to minimize the surgical blood loss, thereby decreasing the need for blood transfusion. By providing a clear surgical field they also decrease the duration of surgery. Most important need in microscopic surgeries is provision of a clear vision.

DEFINITION:

Defined as either of the following [6]

- Reduction of the systolic blood pressure to 80-90mmHg
- Reduction of mean arterial pressure (MAP) to 50-65 mmHg
- 30% reduction of baseline MAP

INDICATIONS:

- Oromaxillofacial surgery
- Endoscopic sinus microsurgery
- Middle ear microsurgery
- Spinal surgery
- Neuro surgery
- Major orthopaedic surgery
- Prostatectomy
- Liver transplant surgery

CONTRAINDICATIONS:

- Congenital heart disease
- Severe anemia
- Coronary artery disease
- Congestive heart failure
- Poorly controlled hypertension
- Increased intracranial pressure
- Significant cerebro-vascular disease
- Extremes of age
- Hypovolemia

METHODS USED FOR INDUCING HYPOTENSION

- Physiologic technique
- Pharmacologic technique

PHYSIOLOGIC TECHNIQUES

- Body positioning
- Hemodynamic effects of mechanical ventilation
- Changes in heart rate & circulatory volume

PHARMACOLOGIC TECNIQUES:

Pharmacological agents can generally be divided into two

1. **Inhalational agents**

Commonly used inhalation agents are halothane and isoflurane. The concentration of a volatile anesthetic agent produces a dose dependent decrease in mean arterial pressure. They have negative inotropic and vasodilatory effects.

2. Peripheral vasodilators

The three most commonly used vasodilators are: sodium Nitroprusside (SNP), Nitroglycerin (NTG) and Trimethaphan.

SNP acts as a vascular smooth muscle relaxant and has a rapid onset but brief duration of action. Its primary influence is on arteriolar and venous vessels, but without significant myocardial effects.

NTG reduces blood pressure by relaxing venous smooth muscle and, like SNP, has rapid onset of action but short duration. NTG is less toxic than SNP; however, it is less potent than SNP in its capacity to reduce blood pressure.

Trimethaphan produces hypotension through ganglionic blockade and direct vasodilator properties. It is also short acting and provides tight control of blood pressure.

Beta blocker by decreasing myocardial contractility used for this purpose. The main disadvantage is bronchospasm.

Alpha2 agonists like clonidine and dexmedetomidine are also used for this purpose.

Remifentanil an opioid receptor agonist is also used for controlled hypotension. They have rapid onset and offset. No need for additional hypotensive agents.

Spinal and epidural anesthesia can also be used to produce controlled hypotension.

CHAPTER III

Functional Endoscopic Sinus Surgery

Functional endoscopic sinus surgery is the primary approach used today for the surgical treatment of chronic sinusitis.

The aim of Functional Endoscopic Sinus Surgery (FESS) is to restore the drainage and aeration of the Para nasal sinuses, while maintaining the natural mucociliary clearance mechanism and seeking to preserve the normal anatomic structures [7, 8].

Imaging advances, increased understanding of the anatomy and the pathophysiology of chronic sinusitis, and image-guided surgery have allowed the surgeons to perform more complex procedures with increased safety.

Indications

Endoscopic sinus surgery is most commonly performed for inflammatory and infectious sinus disease. The most common indications for endoscopic sinus surgery are as follows:

- Chronic sinusitis refractory to medical treatment
- Recurrent sinusitis
- Nasal polyposis
- Antrochoanal polyps
- Sinus mucoceles
- Excision of selected tumors
- Cerebrospinal fluid (CSF) leak closure
- Orbital decompression (e.g., Graves ophthalmopathy)
- Optic nerve decompression
- Dacryocystorhinostomy (DCR)
- Choanal atresia repair
- Foreign body removal
- Epistaxis control

FESS is a delicate and time consuming procedure. It is performed routinely under general anesthesia. Can also be done under local anesthesia.

Anesthesiologists have to plan the technique in such a way that will facilitate the operating team for achieving a bloodless field for better visualization of the intranasal structures and minimize intra operative bleeding, because even minimal bleeding can obstruct the view of the operating endoscope. Hence comes the role of hypotensive anesthesia.

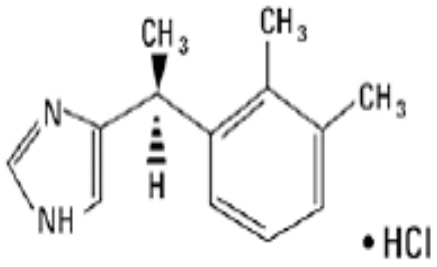
Dexmedetomidine, an imidazole compound, is the pharmacologically active Dextroisomer of medetomidine, and is the most selective central α2 - adrenoceptor agonist available clinically.

Dexmedetomidine offers beneficial pharmacological properties, providing dose dependent sedation, analgesia, sympatholysis and anxiolysis without relevant respiratory depression. The dominant action of adrenoceptor α2 agonists with low and clinically recommended concentrations is hypotension[7]. Now a day Dexmedetomidine has been used for controlled hypotension in middle ear as well as nasal endoscopic surgeries. The advantage of dexmedetomidine over routinely used agent like nitroglycerine is it will not cause reflex tachycardia.

CHAPTER IV

PHARMACOLOGY OF THE STUDY DRUGS

Dexmedetomidine [9]



It is a highly selective α2-adrenergic agonist that produces sedation, hypnosis and analgesia.

History

The initiation for the use of α2 agonists in anesthesia resulted from observations made in patients during anesthesia who were receiving clonidine therapy. Dexmedetomidine was introduced in clinical practice in the United States in 1999. It was approved by the FDA only as a short-term (<24 hours) sedative for mechanically ventilated adult ICU patients.

Dexmedetomidine is now being used off-label outside of the ICU in various settings.

Pharmacological profile

It is a highly selective α2-adrenergic agonist. It shows a high ratio of specificity for the α2 receptor (α2/α1 1600:1) compared with clonidine (α2/α1 200:1), making it a complete α2 agonist Dexmedetomidine belongs to the imidazole subclass of α2 receptor agonists, similar to clonidine. It is freely soluble in water.

Metabolism and Pharmacokinetics

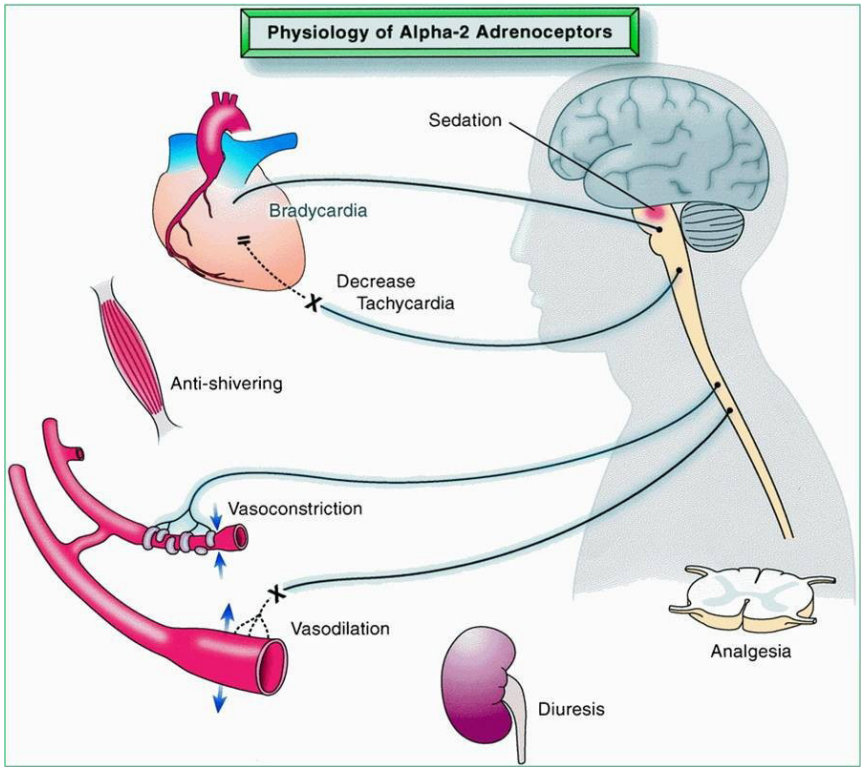
Dexmedetomidine has rapid redistribution. The half life is 6min. Dexmedetomidine is 94% protein bound and its concentration ratio between whole blood and plasma is 0.66.

It is extensively metabolized in the liver. It undergoes conjugation (41%), n-methylation (21%), or hydroxylation followed by conjugation. The inactive metabolites excreted in urine and feces.

The elimination half-life of Dexmedetomidine is 2 to 3 hours, with a context- sensitive half-time ranging from 4 minutes after a 10-minute infusion to 250 minutes after an 8-hour infusion. No accumulation after infusions 12-24 h. Pharmacokinetics similar in young adults and elderly.

Pharmacology

It is a selective α2- adrenoreceptor agonist. Its action is unique and different. Three subtypes of α2 adrenoreceptors have been described in humans: α2A, α2B, and α2C .The α2A adrenoreceptors are present in the periphery where as α2B, and α2C are in the brain and spinal cord. Postsynaptically located α2 adrenoreceptors produce vasoconstriction where as presynaptic α2 adrenoreceptors inhibit the release of norepinephrine, potentially attenuating the vasoconstriction.



The overall response to α2 adrenoreceptor agonists is related to the stimulation of α2 adrenoreceptors located in the CNS and spinal cord. The α2 agonist produces their sedative-hypnotic effect by an action on α2 receptors in the locus caeruleus and an analgesic action at α2 receptors within the locus caeruleus and within the spinal cord.

Effects on the Central Nervous System

Sedation

The α2 agonists act through the endogenous sleep-promoting pathways to exert their sedative effect.

It produces unique sedative quality - someone be clinically sedated yet arousable. Patients sedated, remaining so when unstimulated. But when stimulated they are arousable, alert, and able to respond without becoming uncomfortable.

It‘s also observed that they would quickly return to their sleep-like state.

This characteristic allows for “daily wake up” tests to be done in a safe fashion.

Despite sound levels of sedation with dexmedetomidine, there is limited respiratory depression, providing wide safety margins.

Analgesia

The analgesic effects of dexmedetomidine are complex. Alpha2 agonists do have an analgesic effect when injected via the intrathecal or epidural route. The primary site of analgesic action is thought to be the spinal cord. Systemic use of dexmedetomidine shows narcotic sparing. In the postoperative ICU setting, narcotic requirements were reduced by 50% when patients were receiving a dexmedetomidine.

In human pain studies, the results of systemically administered

Dexmedetomidine are inconsistent. Modest reductions in pain were observed. In the clinical setting, when pain is likely to occur, if dexmedetomidine is to be used, the addition of a narcotic seems warranted.

Other Central Nervous System Effects

Dexmedetomidine in animal models of incomplete cerebral ischemia and reperfusion reduces cerebral necrosis and improves neurologic outcome by reducing the intracerebral catecholamine outflow and the reduction of the excitatory neurotransmitter glutamate during injury.

Dexmedetomidine also is able to reduce muscle rigidity after high-dose opioid administration.

Effects on the Respiratory System

Dexmedetomidine at concentrations producing significant sedation reduces minute ventilation, but retains hypercapnic ventilatory response. The changes in ventilation appeared similar to those observed during natural sleep. Dexmedetomidine has been implicated in blocking histamine-induced bronchoconstriction in dogs.

Effects on the Cardiovascular System

The basic effects of α2 agonists on the cardiovascular system are decreased heart rate; decreased systemic vascular resistance; and indirectly decreased myocardial contractility, cardiac output, and systemic blood pressure.

The hemodynamic effects of a bolus of Dexmedetomidine in humans have shown a biphasic response- an initial increase in blood pressure (22%) and decrease in heart rate (27%) from baseline that occurred at 5 minutes after injection (probably due to the vasoconstrictive effects of dexmedetomidine when stimulating peripheral α2 receptors). Heart rate return to baseline by 15 minutes, and blood pressure decrease 15% below baseline by 1 hour.

The incidence of hypotension and bradycardia may be related to the administration of a loading dose. Omitting the loading dose or not giving more than 0.4 μg/kg reduces the incidence of hypotension. Giving the loading dose over 20 minutes also minimizes the transient hypertension.

Dosage and administration

Dexmedetomidine is supplied in a 2-mL ampoule, 100 mcg/ml.

Dexmedetomidine must be diluted in 0.9% sodium chloride to achieve the required concentrations prior to administration. To prepare the infusion, withdraw 2 ml of dexmedetomidine and add to 48 ml of 0.9% sodium chloride injection to a total of 50 ml. The target concentration is 4 μg /ml.

Loading dose -0.5μg-1μg/kg over 10 min.

Maintenance -0.3μg-0.7μg/kg/hr.

Uses

Dexmedetomidine has been approved as a short-term sedative for adult intubated patients in the ICU. Given its well-documented beneficial effects of anxiolysis, sedation, analgesia, and sympatholysis with minimal respiratory depression, it has been used in various other clinical scenarios.

1. Intensive Care Unit

Dexmedetomidine has following advantages for sedation in mechanically ventilated postoperative patients.

Decreased requirement for opioids (>50%) when dexmedetomidine is used for sedation compared with propofol or benzodiazepines.

Providing adequate sedation with minimal respiratory depression—can be used when weaning patients from the ventilator.

Dexmedetomidine has been successfully used in the treatment of withdrawal of narcotics, benzodiazepines, alcohol, and recreational drugs

2. Anesthesia

a) As a premedicant, dexmedetomidine, at IV doses of 0.33 to 0.67 μg/kg given 15 minutes before surgery, seems efficacious, while minimizing the cardiovascular side effects of hypotension and bradycardia. Within this dose range dexmedetomidine reduces the requirement of thiopentone, volatile anesthetics and attenuates hemodynamic response to endotracheal intubation.

b) IM injection (2.5 μg/kg) with or without fentanyl 45 to 90min before surgery provide angiolysis, reduced response to intubation, smaller volatile anesthetic requirements, and decreased incidence postoperative shivering.

c) Dexmedetomidine is used as a premedication 10 minutes before general surgery for cataract removal, intraocular pressure is decreased (33%), catecholamine secretion is reduced, perioperative analgesic requirements are less, and recovery is more rapid.

d) Dexmedetomidine used for securing the airway with a fiberoptic intubation

e) Dexmedetomidine has been used for sedation for monitored anesthesia care in gynecological, urological, burns patients, trauma patients, pediatric patients[10], and in obese, OSA patients.

f) Sedation during regional anesthesia.

g) Dexmedetomidine also useful as anesthetic adjuvant in Bariatric surgery, Sleep apnea patients, Craniotomy aneurysm, AVM [hypothermia] ,Cervical spine surgery, Off-pump CABG, Vascular surgery, Thoracic surgery, Conventional CABG, Spine surgery, evoked potential study, Head Injury, Burns, Trauma.

h) Dexmedetomidine has also been found to be an effective drug for premedication before i.v regional anesthesia[11]as it reduces patient anxiety, sympathoadrenal responses, and opioid analgesic requirements

Contraindications

• Infusion over 24 hours.

• In obstetric procedures, cesarean section deliveries, as the safety has not been studied.

• Patients with pre-existent severe bradycardia and related bradydysrhythmias (e.g., advanced heart block).

• Patients with impaired ventricular functions (ejection fraction <30%).

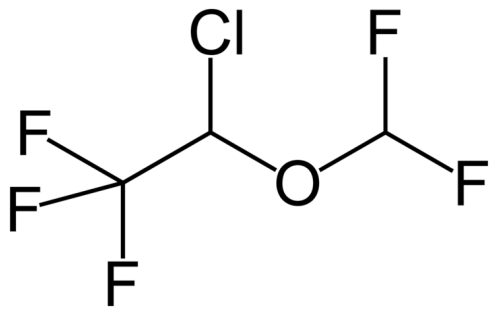
• Patients who are hypovolemic or hypotensive.

• Patient with raised intracranial tension.

Antidote

All effects of dexmedetomidine could be antagonized easily by administering the alpha 2-adrenoceptor antagonist atipamezole [12].

ISOFLURANE



Isoflurane (1-chloro-2, 2, 2-trifluoroethyl difluoromethyl ether) is halogenated ether used for inhalation anaesthesia.

Physical properties[13]

-Molecular weight 184 g

-Boiling point (at 1 atm): 48.5 °C

-Density (at 25 °C): 1.496 g/mL

-MAC: 1.17

-Vapour pressure: 238 mm Hg 31.7 kPa (at. 20°C)

-Blood: Gas Partition coefficient: 1.46

-Oil: Gas Partition coefficient: 91

Isoflurane is a non inflammable volatile annesthetic with a pungent ethereal odour.Although it is a chemical isomer of enflurane, it has different physicochemical properties.

Mechanism of Action of Inhaled Anesthetics [14]

Inhaled anesthetics act in different ways at the level of the central nervous system. They may disrupt normal synaptic transmission by interfering with the release of neurotransmitters from presynaptic nerve terminal (enhance or depress excitatory or inhibitory transmission), by altering the re-uptake of neurotransmitters, by changing the binding of neurotransmitters to the post synaptic receptor sites, or by influencing the ionic conductance change that follows activation of the post-synaptic receptor by neurotransmitters. Both, pre- and postsynaptic effects have been found. The high correlation between lipid solubility and anesthetic potency suggests that inhalation anesthetics have a hydrophobic site of action. Inhalation agents may bind to both membrane lipids and proteins.

The Meyer-Overton theory describes the correlation between lipid solubility of inhaled anesthetics and MAC and suggests that anesthesia occurs when a sufficient number of inhalation anesthetic molecules dissolve in the lipid cell membrane. The Meyer-Overton rule postulates that the number of molecules dissolved in the lipid cell membrane and not the type of inhalation agent causes anesthesia.

Mullins expanded the Meyer-Overton rule by adding the so-called Critical Volume Hypothesis. He stated that the absorption of anesthetic molecules could expand the volume of a hydrophobic region within the cell membrane and subsequently distort channels necessary for sodium ion flux and the development of action potentials necessary for synaptic transmission.

The protein receptor hypothesis postulates that protein receptors in the central nervous system are responsible for the mechanism of action of inhaled anesthetics. This theory is supported by the steep dose response curve for inhaled anesthetics.

Another theory describes the activation of Gamma-Amino Butyric Acid (GABA) receptors by the inhalation anesthetics. Volatile agents may activate GABA channels and hyperpolarize cell membranes. In addition, they may inhibit certain calcium channels and therefore prevent release of neurotransmitters and inhibit glutamate channels. Volatile anesthetics share therefore common cellular actions with other sedative, hypnotic or analgesic drugs.

Pharmacokinetics:

Uptake and distribution of inhaled anesthetics [14]

A series of partial pressure gradients, beginning at the vaporizer of the anesthetic machine, continuing in the anesthetic breathing circuit, the alveolar tree, blood, and tissue will ensure the forward movement of the gas. The principal objective of that movement is to achieve equal partial pressures on both sides of each single barrier. The alveolar partial pressure governs the partial pressure of the anesthetic in all body tissues; they all will ultimately equal the alveolar partial pressure of the gas. After a short period of equilibration the alveolar partial pressure of the gas equals the brain partial pressure. Alveolar partial pressure can be raised by increasing minute ventilation, flow rates at the level of the vaporizer and by using a non-rebreathing circuit.

Isoflurane has a blood/gas partition coefficient of 1.4. This means that if the gas is in equilibrium the concentration in blood will be 1.4 times higher than the concentration in the alveoli. A higher blood gas partition coefficient means a higher uptake of the gas into the blood and therefore a slower induction time. It takes longer until the equilibrium with the brain partial pressure of the gas is reached.

A higher cardiac output removes more volatile anesthetic from the alveoli and therefore lowers the alveolar partial pressure of the gas. The agent might be rapidly distributed within the body but the partial pressure in the arterial blood is lower. It will take longer for the gas to reach equilibrium between the alveoli and the brain. Therefore, a high cardiac output prolongs induction time.

The alveolar to venous partial pressure difference reflects tissue uptake of the inhaled anesthetics. Isoflurane has a brain/blood coefficient of 1.6 meaning that if the gas is in equilibrium the concentration in the brain will be 1.6 times higher than the concentration in the blood. All inhalation anesthetics have high fat/blood partition coefficients. This means that most of the gas will bind to fatty tissue as times goes by. The partial pressure of the gas in fatty tissue will rise very slowly. Inhalation anesthetics stored in such tissue in obese patients may delay awakening at the end of anesthesia.
Isoflurane shows very low solubility in blood and body tissues. Thus its partial pressure (concentration) in alveolar gas or arterial blood rises to 50% of the inspired partial pressure (concentration) within 4-8 minutes of the start of its inhalation, and to 60% within 15 minutes.

Throughout maintenance of anesthesia, a high proportion of the Isoflurane inspired is eliminated by the lungs. When administration is stopped and inspired concentration becomes zero, the bulk of the remaining Isoflurane is eliminated unchanged from the lungs. In keeping with its low solubility, recovery from Isoflurane anesthesia in man is rapid.

Effect on cardio vascular system

Isoflurane causes minimal cardiac depression in vivo. Cardiac output is maintained by a rise in heart rate due to partial preservation of carotid baroreflexes. Isoflurane dilates coronary arteries, particularly if its concentration is abruptly increased, although it is not nearly as potent a dilator as nitroglycerine or adenosine.

Effect on respiratory system

Respiratory depression during isoflurane anesthesia resembles that of other volatile anesthetics, except that tachypnoea is less pronounced. The net effect is a more pronounced fall in minute ventilation. Despite a tendency to irritate upper airway reflexes, isoflurane is considered a good bronchodilator, but may not be as potent a bronchodilator as halothane.

Effect on renal system

Like other inhalational anesthetic agents, isoflurane decreases renal blood flow, glomerular filtration rate and urinary output transiently but hepatic oxygen supply is better maintained with isoflurane than halothane or enflurane.

Metabolism

Biotransformation of Isoflurane is significantly less than that of Enflurane or Halothane. Human biotransform a small fraction of Isoflurane administered. In man about 0.2% of the Isoflurane administered, is evident as recoverable metabolites (fluoride and organic fluorine), with approximately 50% of these excreted in the urine, the principal metabolite being trifluoracetic acid. Although serum fluoride levels may rise, nephrotoxicity is extremely unlikely even in the presence of enzyme inducers. Prolonged sedation (>24 hours at 0.1-0.6%) of critically ill patients has resulted in elevated plasma fluoride levels without evidence of renal impairment.

USES

Induction:

As Isoflurane has a mild pungency, inhalation should usually be preceded by the choice of a short-acting barbiturate, or other intravenous induction agent, to prevent coughing.

Alternatively, Isoflurane with oxygen or an oxygen/nitrous oxide mixture may be administered. It is recommended that induction with Isoflurane be initiated at a concentration of 0.5%. Concentrations of 1.5-3.0% usually produce surgical anesthesia in 7-10 minutes. Blood pressure decreases during induction but this may be compensated by surgical stimulation.

Maintenance:

Adequate anesthesia for surgery may be sustained with an inspired Isoflurane concentration of 1.0% - 2.5% in an oxygen/70% nitrous oxide mixture. Additional inspired Isoflurane (0.5% - 1%) will be required with lower nitrous oxide levels, or when Isoflurane is given with oxygen alone or air/oxygen mixtures. Blood pressure decreases during maintenance anesthesia in relation to the depth of anesthesia. That is, blood pressure is inversely related to the Isoflurane concentration. Provided there are no other complicating factors this is probably due to peripheral vasodilatation.

Cardiac rhythm remains stable. Excessive falls in blood pressure may be due to the depth of anesthesia and in such circumstances can be corrected by reducing the inspired Isoflurane concentration.

Controlled hypotension

Used to produce controlled hypotension either alone or in combination with other drugs.

In studies on humans and animals, isoflurane decreased blood pressure by decreasing SVR, whereas cardiac output was maintained constantly at clinically relevant concentrations of the anesthetic[15]

In healthy young people, 2 to 3 % isoflurane decreases MAP by reducing SVR.

In older or chronically hypertensive patients, similar concentrations of isoflurane may also decrease cardiac output. For these individuals, combining a moderate concentration of isoflurane with agents that tend to maintain cardiac output would be more appropriate than using high concentrations of isoflurane alone.

Isoflurane appears to offer certain advantages over other techniques commonly used to induce hypotension[16] At lower cerebral perfusion pressures (<30 mmHg), the cerebral metabolic rate for oxygen was better

preserved, suggesting cerebral protection. Isoflurane also favorably influenced the global cerebral oxygen supply/demand ratio in humans having a MAP of 50 mmHg[17]

Recovery:

The concentration of Isoflurane can be reduced to 0.5% at the start of closing the operation wound, and then to 0% at the end of surgery. After discontinuation of all anesthetics, the airways of the patient should be ventilated several times with oxygen 100% until complete recovery.

CHAPTER V

REVIEW OF LITERATURE

This study was designed to evaluate the effect of Dexmeditomidine infusion on the requirement of Isoflurane to maintain mean arterial pressure of 60-70 mmHg. Literature was reviewed to analyze the existence of similar studies.

1. Mohammad Maroof, et al,[18] in their study on 'Dexmedetomidine Is a Useful Hypotensive Adjunct during Middle Ear Surgery under General Anesthesia' recruited 42 ASA I or II adult patients scheduled for elective Middle Ear Surgery and were randomly divided into 2 equal groups.

Group-I: Received 10-15 min before induction of anesthesia, placebo bolus and infusion of saline at a rate similar to DEX in group-II.

Group-II: Received 10-15 min prior to induction of anesthesia 1 μg/ kg iv bolus DEX diluted in 10 ml of normal saline over 10 min. anesthesia was maintained with 60% nitrous oxide + 40% oxygen + isoflurane titrated to achieve a mean arterial pressure [MAP] 30% below the control value (value taken after premedication). Isoflurane and DEX/ SAL infusion was stopped 8-10 min prior to end of surgery. They analyzed the isoflurane requirement, quality of surgical field , awakening time and concluded that DEX infusion aids in achieving a targeted reduction in MAP, better blood less field, faster

awakening and reduced Isoflurane requirement in patients undergoing middle ear surgery.

2. Durmus M, et al, [19] in their study on 'Effect of dexmedetomidine on bleeding during tympanoplasty or septorhinoplasty' recruited 40 adult patients randomly assigned them to receive either a bolus dexmedetomidine or placebo before induction of anesthesia followed by infusion. They maintained mean arterial pressure around 60-80mmHg. They analyzed Perioperative mean arterial pressure, heart rate, time to extubation and time to awakening. They found that propofol dose required for induction, intraoperative fentanyl, isoflurane requirement and bleeding were low in dexmedetomidine group than control and concluded that dexmedetomidine is a useful adjuvant to decrease bleeding when a bloodless surgical field is requested.

3. Guldem Turan, et al[20] in their study on 'Comparison of dexmedetomidine, Remifentanil and Esmolol in Controlled Hypotensive Anesthesia' recruited 70 adult patients undergoing tympanoplasty into three groups. Group D (n=26) Dexmedetomidine 1 µg /kg (10 min) loading, 0.2-0.7µg/kg/h infusion Group R (n=21) Remifentanil 0.2-0.5 µg/ kg/min Group E (n=23) Esmolol 500 µg/kg(1min) loading, 50-300µg /kg/ min infusion. They maintained anesthesia with desflurane 3-6%

They analyzed the quality of surgical field, Spontaneous eye opening time, extubation time, verbal response time, cooperation and orientation time and concluded that dexmedetomidine, remifentanil and esmolol may be used for controlled hypotension during tympanoplasty operations in respect of intraoperative bleeding, recovery and adverse effects.

4.Hilal Ayoglu, et al [21] in their study on 'Effectiveness of dexmedetomidine in reducing bleeding during septoplasty and tympanoplasty operations ' recruited 80 adult patients undergoing tympanoplasty and septoplasty and were divided them into four groups. Dexmedetomidine (D) was administered to Group SD(20) and Group TD(20) first as a bolus dose of one μg /kg, then intraoperative maintenance of dexmedetomidine 0.7 μg/kg/ hour. Groups S(20) and T(20) (controls) were given identical amount of saline. They used thiopentone 6mg/kg , rocuronium 0.6mg/kg for induction and sevoflurane for maintenance. They were analyzed intraoperative blood loss,hemodynamic parameters, fentanyl requirements and concluded that dexmedetomidine reduces bleeding, intraoperative fentanyl consumption and improve visibility of the field during septoplasty.Dexmedetomidine also significantly decrease fentanyl need in tympanoplasty but the decrease in intraoperative bleeding was not significant.

5. Iclal Ozdemir Kol, et al, [22] in their study on 'Controlled Hypotension with Desflurane Combined with Esmolol or Dexmedetomidine During Tympanoplasty in Adults' recruited 48 ASA I & II adult patients into two groups (Esmolol and Dex). Esmolol group a loading dose of esmolol was infused intravenously over 1 minute at 1 mg/kg, followed by a maintenance rate of 0.4 to 0.8 mg/kg/h. In the dexmedetomidine group, a loading dose of dexmedetomidine was infused intravenously over 10 minutes at a rate of 1 μg/kg, followed by a maintenance rate of 0.4 to 0.8 μg/kg/h. The infusion rates were then titrated to maintain mean arterial pressure (MAP) of 65 to 75 mm Hg. General anesthesia was maintained with desflurane 4% to 6%. They analyzed the amount of blood loss in the surgical field, recovery time and tolerability in adult patients. They concluded that both esmolol and dexmedetomidine, combined with desflurane, provided an effective and well-tolerated method for achieving a bloodless surgical field with controlled hypotension in these patients undergoing tympanoplasty. Esmolol was associated with significantly shorter extubation and recovery times and significantly less postoperative sedation compared with dexmedetomidine.

6.Farah Nasreen , et al, [23] in their study on Dexmedetomidine used to provide hypotensive anesthesia during middle ear surgery recruited 42 patients into two groups (Dex and NS).Group I received NS equal to dexmedetomidine and Group II received 1mcg/kg dex as bolus 10-15 min before induction followed by infusion of 0.5mcg/kg/hr. They titrated halothane to maintain mean arterial pressure 30% below the control value. They observed that a statistically significant reduction in the percentage of halothane requirement in group II (1.3 ± 0.4%) in comparison to group I (3.1 ± 0.3%). Patients receiving DEX infusion had a better surgical field. The mean awakening time was significantly reduced in patients of Group II (9.1 ± 2.7 min) when compared to patients of Group I (12.8 ± 2.2 min) and concluded that DEX can be safely used to provide hypotensive anesthesia during middle ear surgery.

7. Lawrence CJ,et al, [24] in their study on 'Effects of a single pre-operative dexmedetomidine dose on isoflurane requirements and peri-operative haemodynamic stability ' placebo-controlled study investigated the effect of a single pre-induction intravenous dose of dexmedetomidine2 micrograms.kg-1 on anesthetic requirements and peri-operative hemodynamic stability in 50 patients undergoing minor orthopaedic and general surgery. The mean (SD) intra-operative isoflurane concentration was

lower in the dexmedetomidine-treated patients than controls (0.01 (0.03)% compared to 0.1 (0.1)%; p = 0.001) .They found that the haemodynamic response to tracheal intubation and extubation was reduced in the dexmedetomidine group. The intra-operative heart rate variability; postoperative analgesic and anti-emetic requirements and peri-operative serum catecholamine concentrations were also lower in the dexmedetomidine group. Hypotension and bradycardia occurred more frequently after dexmedetomidine.

8. Khan ZP, et al, [25] in their study on 'Effects of dexmedetomidine on isoflurane requirements in healthy volunteers' concluded that dexmedetomidine decreases isoflurane requirements in a dose-dependent manner and reduced heart rate, systolic and diastolic arterial pressures. Sedation and slight impairment of cognitive function persisted for several hours after anesthesia and the end of infusion of dexmedetomidine. Isoflurane did not appear to influence the pharmacokinetics of dexmedetomidine.

9.Aantaa R, et al, [26] in their study on ' Reduction of the minimum alveolar concentration of isoflurane by dexmedetomidine ' recruited 49 patients scheduled for abdominal hysterectomy and randomly allocated to receive either a placebo infusion (n = 16) or a two-stage infusion of

dexmedetomidine with target plasma concentration of 0.3 ng/ml (n = 17) or 0.6 ng/ml (n = 16). The study drug infusion was commenced 15 min before induction. The end-tidal concentration of isoflurane for each patient was predetermined according to the "up-down" method of Dixon, and it was maintained for at least 15 min before the patient's response to skin incision was assessed. They found the MAC of isoflurane was 0.85% end-tidal in the control group, 0.55% end-tidal with the low dose of dexmedetomidine, and 0.45% end-tidal with the high dose of dexmedetomidine.

10. Bayazit Dikmen, et al, [27] in their study on 'Dexmedetomidine for Controlled Hypotension in Middle Ear Surgery with Low-Flow Anesthesia ' Forty patients undergoing middle ear surgery were studied. In Group D (n=20), Dexmedetomidine (0.1µg/kg/min for 10 minutes) was administered before induction and continued with a rate between 0.2-0.7 µg/kg/h and Group S(n=20) received normal saline with a rate of 50 ml.h-1. Infusions were stopped at the end of microsurgery. Anesthesia was induced with thiopental and vecuronium bromide. Maintenance of anesthesia was achieved by 1.5 % isoflurane delivered in mixture of O2 and N2O 4.4 L.min-1 for 10 min and then flow rate was reduced to 1 L.min-1 and isoflurane concentration was increased to 2 %. Haemodynamic parameter, quality of the surgical field and surgeon satisfaction were evaluated. They

concluded that Dexmedetomidine was effective in inducing consistent and sustained controlled hypotension in low-flow anesthesia during middle ear microsurgery.

11.Richa F, et al, [28] in their study on 'Comparison between dexmedetomidine and remifentanil for controlled hypotension during tympanoplasty' recruited 24 patients into two groups (Dex and Remifentanil). They found that infusion of dexmedetomidine, at the doses (0.4-0.8 mcg/ kg/hr) used in this study, was less effective than remifentanil in achieving controlled hypotension, good surgical field exposure condition and surgeon's satisfaction during tympanoplasty.

12. Berrin Isik, et al, [29] in their study on 'The Effects of Adrenergic Receptor Agonist Dexmedetomidine on Hemodynamic Response in Direct Laryngoscopy ' recruited 40 patients scheduled for direct laryngoscopy under general anesthesia. The patients were randomly divided into two groups, Intramuscular 0.05 μg /kg midazolam (Group M) or intravenous 1 μg /kg dexmedetomidine (Group D) was applied. Heart Rate and mean arterial pressure (MAP) were measured before premedication and noted down as control values. Preoperative hemodynamic parameters, recovery times and sedation levels of both groups were compared. They concluded that dexmedetomidine premedication in direct laryngoscopy procedures

controls hypertension and tachycardia more efficiently without prolonged recovery time than midazolam premedication.

13.Hulaya Basar, et al, [30] in their study on 'The effects of preanesthetic, single-dose dexmedetomidine on induction, hemodynamic, and cardiovascular parameters ' recruited 40 patients scheduled for elective cholecystectomy and divided into two groups to receive 0.5 μg kg^{-1} dexmedetomidine (group D, n = 20) or saline solution (group C, n = 20). Anesthesia was induced with thiopental sodium and vecuronium, and anesthesia was maintained with 4% to 6% desflurane. They analyzed Mean arterial pressure (MAP), heart rate (HR), ejection fraction (EF), end-diastolic index (EDI), cardiac index (CI), and stroke volume index (SVI) were recorded at 10-minute intervals. The times for patients to "open eyes on verbal command" and postoperative Aldrete recovery scores were also recorded. They concluded that a single dose of dexmedetomidine given before induction of anesthesia decreases thiopental requirements without serious hemodynamic effects or any effect on recovery time.

14. Goksu.S,et al, [31] in their study on 'Effects of dexmedetomidine infusion in patients undergoing functional endoscopic sinus surgery under local anesthesia ' Sixty-two patients who were planned to undergo functional endoscopic sinus surgery under local anesthesia were included in this study

and divided into Dex and NS groups. Dexmedetomidine bolus intravenous infusion (an initial loading dose of 1 μg kg^{-1} given for a 10-min period followed by 0.7 μg/ kg/h) was administered to the treatment group. They concluded that dexmedetomidine provides analgesia, adequate sedation and surgical comfort without adverse effects for patients undergoing functional endoscopic sinus surgery under local anesthesia.

15. Damla Guclu Guven, et al, [32] in their study on 'Evaluation of Outcomes in Patients given Dexmedetomidine in Functional Endoscopic Sinus Surgery ' Forty patients who underwent FESS were enrolled in this study. In the DEX group, conscious sedation was induced with an infusion of 1 μg/kg of DEX bolus, followed by an infusion of DEX at 0.2 μg/kg per hour. A control group was given identical amounts of saline solution. During the procedure, hemodynamic data were recorded. The patients evaluated their pain on a visual analog scale (VAS). Intraoperative bleeding was rated on a 6-point scale for evaluation of operative field visibility. They observed that the intraoperative bleeding, hemodynamic stability, and VAS scores were better and the side effects were less frequent in the DEX group.

16. Alp Gurbet , et al, [33] in their study on 'Intraoperative infusion of dexmedetomidine reduces perioperative analgesic requirements ' Fifty women were randomly assigned to two groups. Group D (*n* = 25) received a loading dose of dexmedetomidine 1 μg·kg–1 *iv* during induction of anesthesia, followed by a continuous infusion at a rate of 0.5 μg·kg–1·hr–1 throughout the operation. Group P (*n* = 25) received a volume-matched bolus and infusion of placebo (0.9% saline). They analyzed perioperative hemodynamics and post operative pain score and morphine requirement. They observed that Continuous *iv* dexmedetomidine during abdominal surgery provides effective postoperative analgesia, and reduces postoperative morphine requirements without increasing the incidence of side effects.

17.Uzumcugil F, et al, [34] in their study on 'Comparison of dexmedetomidine-propofol vs. fentanyl-propofol for laryngeal mask insertion ' recruited 52 patients posted for urological procedures divided into two groups. Group F received 1 kg(-1) μg fentanyl (in 10 mL normal saline) and Group D received μg kg(-1) dexmedetomidine. They observed that jaw mobility, coughing or movement and other events such as spontaneous ventilation, breath holding, expiratory stridor and lacrimation. They concluded that Dexmedetomidine, when used before propofol induction

provides successful laryngeal mask insertion comparable to fentanyl, while preserving respiratory functions more than fentanyl

18. Yezdan Firat, et al, [35] in their study on 'The effect of dexmedetomidine on middle ear pressure ' recruited 60 patients into two groups (Dex and NS). The alteration of middle ear pressure values from baseline were analyzed in both groups. In which the differences from the baseline were statistically significant in both groups. They concluded that Dexmedetomidine should be preferred in middle ear surgery requiring good surgical field visibility and normal middle ear pressure.

CHAPTER VI

AIM

To Evaluate the effect of Dexmedetomidine infusion on the requirement of Isoflurane to produce controlled hypotension(mean arterial pressure of 60-70mmHg), quality of bloodless surgical field, duration of surgery and the awakening time in patients undergoing Functional Endoscopic Sinus Surgery(FESS).

CHAPTER VII

MATERIALS AND METHODS

This study was conducted after obtaining approval from ethical committee and patients consent.

Study Design

Prospective randomized control study

Patient selection

50 ASA I Patients age 18-60 years diagnosed having chronic sinusitis scheduled for FESS under general anesthesia were divided into two groups.

Exclusion Criteria

1. Hypertensive patients.

2. H/o Cerebro-vascular accident / Transient ischaemic attack.

3. IHD.

4. Poor respiratory reserve.

5. Significant hepatic or renal disease.

6. Hypersensitivity to study drugs.

7. Patients who are not willing to participate in the study

Materials

1. Perfusor compact-Syringe infusion pump.

2. Inj.Dexmedetomidine 2 ml amp, Normal saline.

3. Disposable 50 ml syringe.

4. Extension tube.

5. Weighing machine.

6. Monitors – ECG, NIBP, SPO2

Methodology

50 patients with the above criteria were divided into two equal groups.

Group D: Received bolus dose of Dexmedetomidine 1 μg /kg over ten min before induction followed by infusion of 0.5 μg /kg/hr. (2ml of dexmedetomidine was diluted with 48ml of NS making a solution of 4 μg /ml)

Group C: Received equal amount of Normal Saline.

Preoperative investigations reports like Hb%, Blood Urea, Serum Creatinine, Platelets, Clotting time, Bleeding time were recorded.

On arriving to the operating room monitors were connected and baseline vital parameters were noted. Two peripheral intravenous line with 18 G IV Cannula one for IV infusion another for study drug were started. Preloading was done with 10 ml/kg of balanced salt soulations.

Premedicated with Inj. Glycopyrrolate 5 mcg/kg +Inj. Fentanyl 2 mcg/kg. The study drugs were started according to the group.

Then anesthesia was induced with Inj.Propofol 2mg/kg + Inj. Vecuronium 0.1 mg/kg and intubated with appropriate size endotracheal tube. Throat packed with saline socked pack.

Anesthesia was maintained with 66% N2O + 33% O2 + IPPV with titrated dose of isoflurane and Vecuronium.

The mean arterial pressure was maintained around 60-70mmHg by titrating the intra operative isoflurane percentage. The isoflurane concentration was recorded every five min and averaged for analysis.

Intra-operative hypotension was managed by

1. IV fluids LR/NS 200 ml
2. Taper down isoflurane
3. Ephedrine 3mg i.v. bolus

Intra-op Tachycardia (HR > 150 bpm) controlled by i.v. Metoprolol 1-5 mg

Intra-op bradycardia (HR < 50 bpm) managed by, 0.3 mg atropine every 2 –3 min till it reached above 60 beats/ min

Intra-op Arrhythmias:

If haemodynamically stable, continue with the study but with close and increased monitoring.

If unstable, abandon hypotension, volume resuscitate and manage accordingly.

Both study drug and isoflurane were stopped 10-15 min prior to end of surgery. Inj. Ondansetron 4mg was given intrapoeratively. The throat pack was removed at the end of the endoscopic procedure.

The residual neuromuscular blockade was reversed with inj.neostigmine 50 mcg/kg+inj. Glcopyrrolate 10mcg/kg and was extubated. The awakening time in min (clearly telling their name) from the time of extubation were recorded.

Patients were observed in the recovery room for nausea & vomiting, sedation score and then monitored in the postoperative ward. Both groups were hemodynamically stable and none showed any adverse reactions like reflex hypertension, nausea and vomiting.

Parameters studied

1. Heart rate

2. Systolic blood pressure

3. Diastolic blood pressure

4. Mean arterial pressure.

5. Requirement of isoflurane percentage

6. Intraoperative problems (hypotension, hypertension, arrythmias, tachycardia, bradycardia and ischemia

7. Duration of surgery.

8. Quality of operating field .

9. Awakening time.

10. Ramsay sedation scale.

Intraoperative surgical field was assessed by using Fromme-Boezaart scale as given below

Surgical field grading: **Fromme –Boezaart scale**

(Evaluation scale for bleeding of surgical field)

Grade O: No bleeding.

Grade 1: Slight bleeding-No suctioning of blood required.

Grade 2: Slight bleeding-Occasional suctioning required. Surgical field not threatened.

Grade 3: Slight bleeding-Frequent suctioning required. Bleeding threatens surgical field a few seconds after suction is removed.

Grade 4: Moderate bleeding- Frequent suctioning required. Bleeding threatens surgical field directly after suction is removed.

Grade 5: Severe bleeding-Constant suctioning required. Bleeding appears faster than can be removed by suction. Surgical field severely threatened and surgery impossible

Postoperative sedation were assessed by following scale

Ramsay Sedation Scale

1 Patient is anxious and agitated or restless, or both

2 Patient is cooperative, oriented and tranquil

3 Patient responds to commands only

4 Patient exhibits brisk response to light glabellar tap or loud auditory stimulus

5 Patient exhibits a sluggish response to light glabellar tap or loud auditory stimulus

6 Patient exhibits no response

Data Management and Analysis

The variables were entered into SPSS, version 15, statistical software for analysis. Statistical analysis was done by using descriptive statistics and cross tabulation. Mean and standard deviation were used to assess changes within and between the two groups. The difference in proportions is tested for statistical significance using non parametric chi-square test for variables measured on nominal scale. For variables measured on a continuous scale, student “t” Test was used. A p value of <0.05 was considered to be statistically significant.

CHAPTER VIII

OBSERVATION AND RESULTS

DEMOGRAPHIC DATA

TABLE I

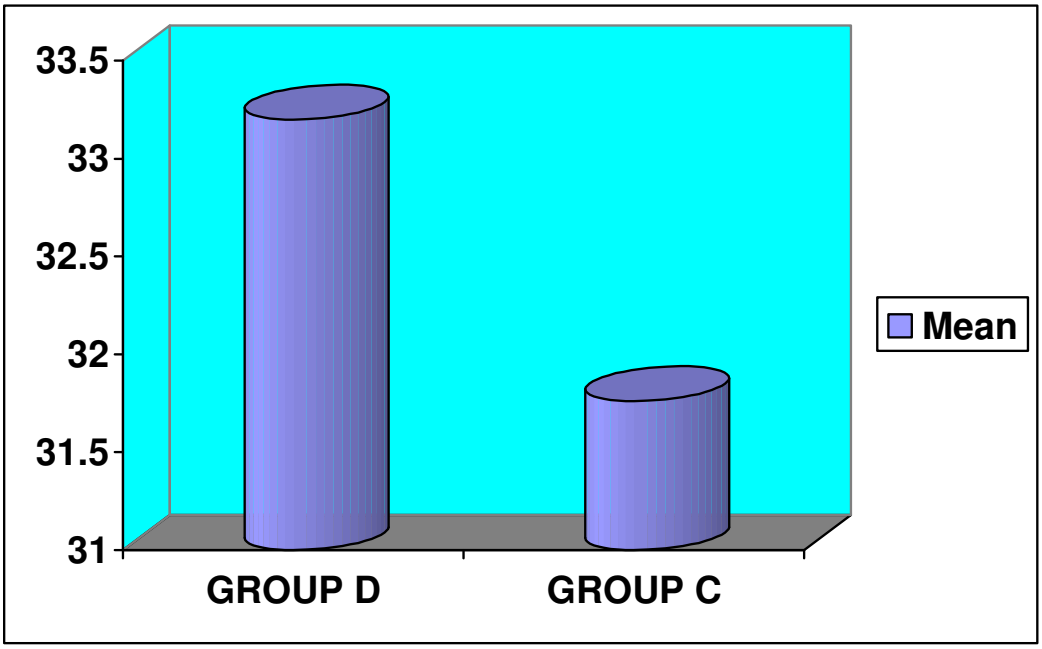
Age distribution

Student "t"test

	Group D	Group C	p-value
No. of cases	**25**	**25**	**0.611**
Mean	**33.20**	**31.76**	
S.D	**9.574**	**10.325**	
Range	**18-53**	**18-55**	

Not statistically significant

AGE DISRIBUTION



The mean age between the comparison groups were almost similar.

The minimum age taken for the study was 18 and the maximum was 55.

TABLE II

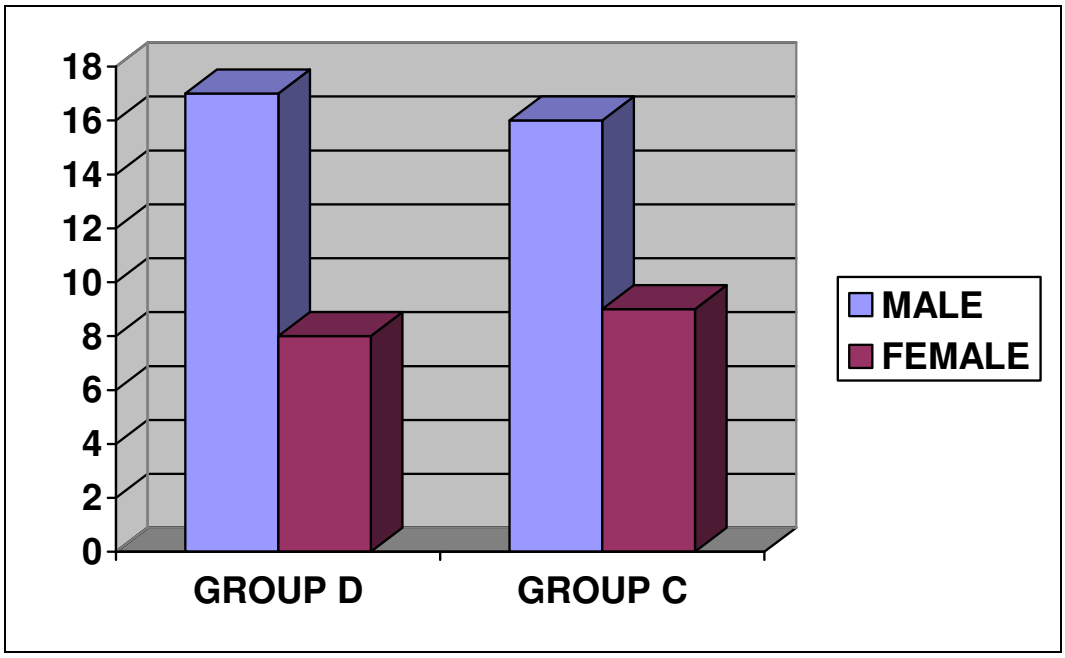
Sex Distribution

Chi-Square Test

	Group D		Group C		p-value
	Nos.	%	Nos.	%	
Male	17	68	16	64	
Female	8	32	9	36	1.000

Not statistically significant

SEX DISTIBUTION



The male preponderance was forthcoming in all the study groups.

However the distribution of sex among the groups was not statistically significant.

TABLE III

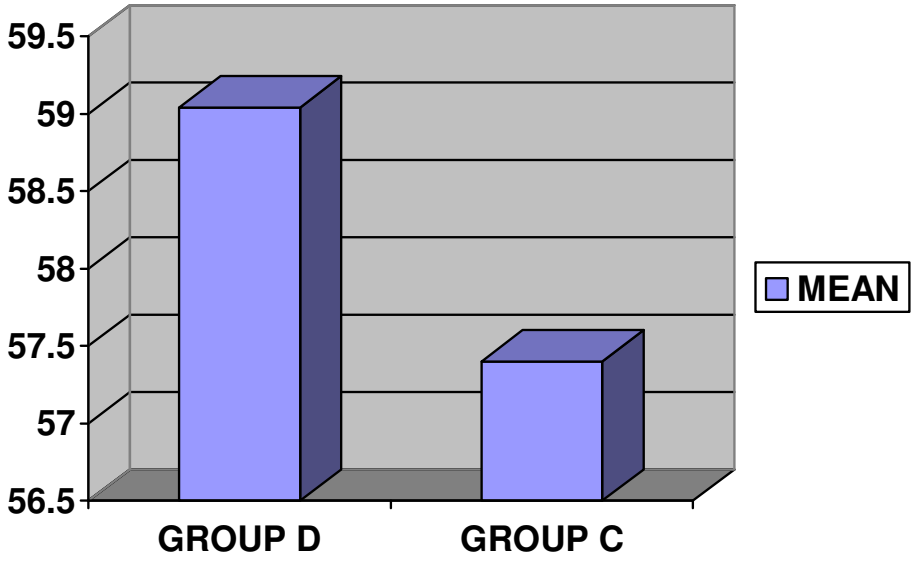
Weight Distribution

Student "t" test

	Group D	Group C	p-value
No. of cases	25	25	0.331
Mean	59.04	57.40	
S.D	6.465	5.284	
Range	45-70	45-65	

Not statistically significant

WEIGHT DISRIBUTION



The mean distribution of cases by weight was observed to be not statistically significant between the two groups.

TABLE IV

Intraoperative hemodynamic parameters

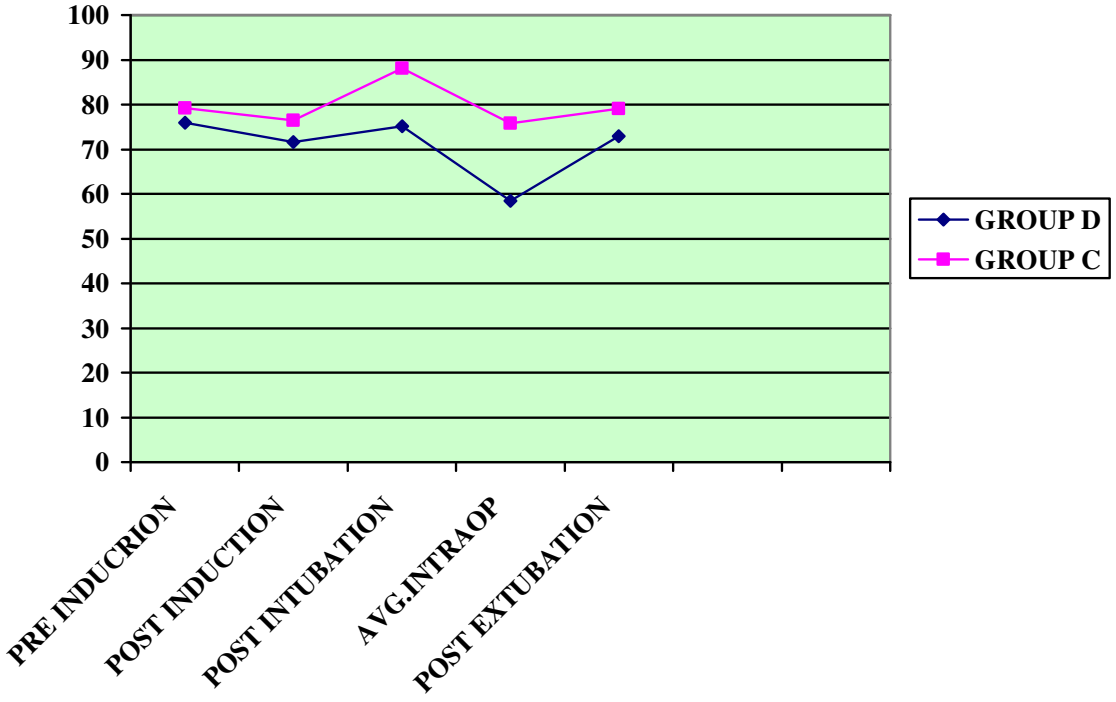
Student "t" test

Parameter	Group D	Group C	p-value
Heart Rate			
Pre Induction	**75.92±5.787**	**79.24±8.695**	**0.119**
Post Induction	**71.68±6.830**	**76.48±9.417**	**0.045***
Post Intubation	**75.12±5.761**	**88.04±7.618**	**0.001***
Avg. Intraop	**58.44±2.873**	**75.84±6.472**	**0.001***
Post Extubation	**72.88±5.231**	**79.04±11.681**	**0.020***
SBP			
Pre Induction	**122.28±8.532**	**121.28±8.824**	**0.639**
Post Induction	**105.96±10.597**	**114.88±13.486**	**0.012***
Post Intubation	**100.84±12.701**	**117.56±12.145**	**0.001***
Avg. Intraop	**91.16±1.864**	**93.40±3.440**	**0.006***
Post Extubation	**116.88±9.528**	**124.00±10.194**	**0.014***
DBP			
Pre Induction	**81.36±6.376**	**78.72±7.220**	**0.177**
Post Induction	**69.44±7.896**	**71.60±11.365**	**0.439**
Post Intubation	**65.04±8.039**	**78.40±11.236**	**0.001***
Avg. Intraop	**58.80±1.581**	**61.08±2.499**	**0.001***
Post Extubation	**77.24±8.686**	**80.76±9.701**	**0.183**
MAP			
Pre Induction	**95.00±6.333**	**92.85±7.270**	**0.271**
Post Induction	**81.61±8.058**	**86.03±11.064**	**0.113**
Post Intubation	**76.97±9.217**	**91.45±11.066**	**0001***
Avg. Intraop	**69.72±1.400**	**71.80±2.566**	**0.001***
Post Extubation	**90.45±8.654**	**95.17±9.385**	**0.071**

*statistically significant

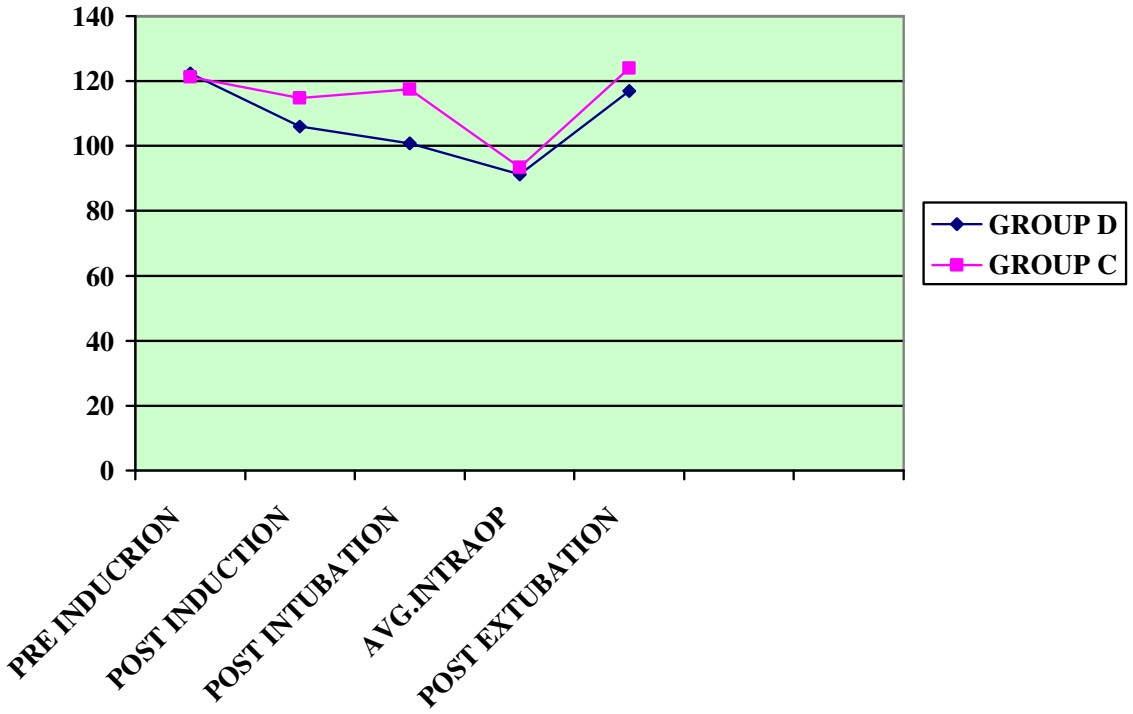
Intraoperative Hemodynamic parameters

Heart Rate



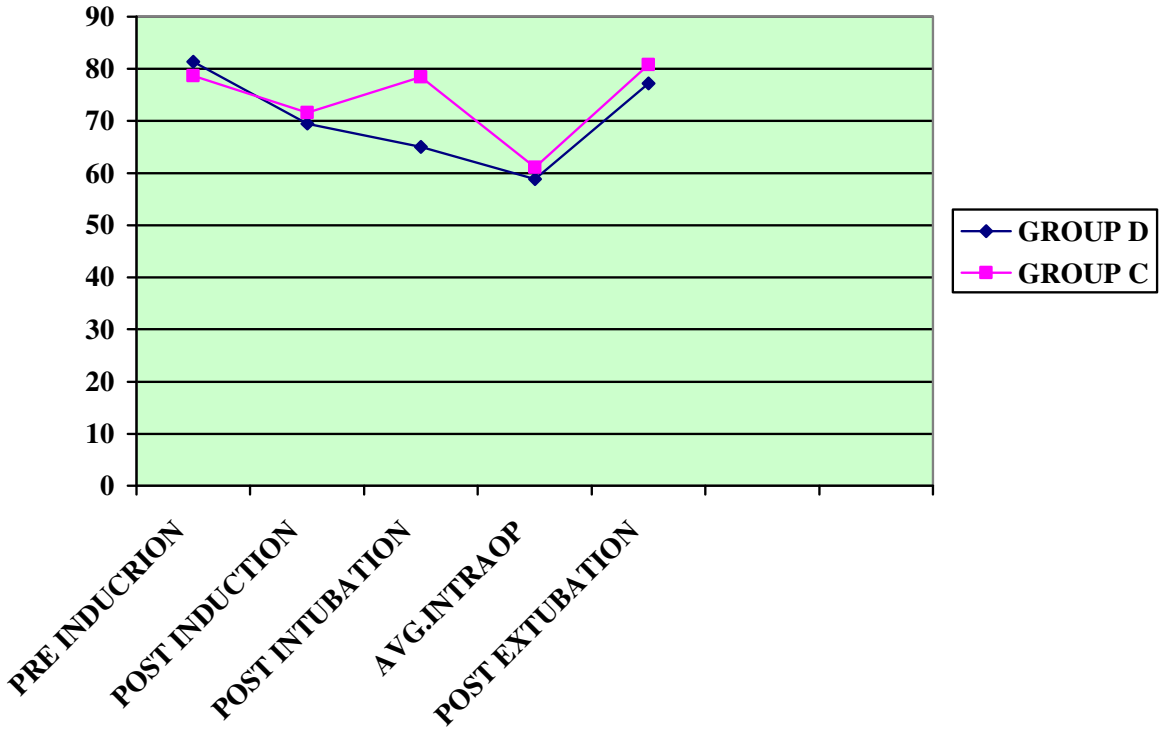
Pre induction heart rate was almost similar. No statistical difference (p-0.119). But the heart rate after induction, after intubation and during intraoperative period was statistically significant which was lower in group D compared to group C.

Systolic Blood Pressure



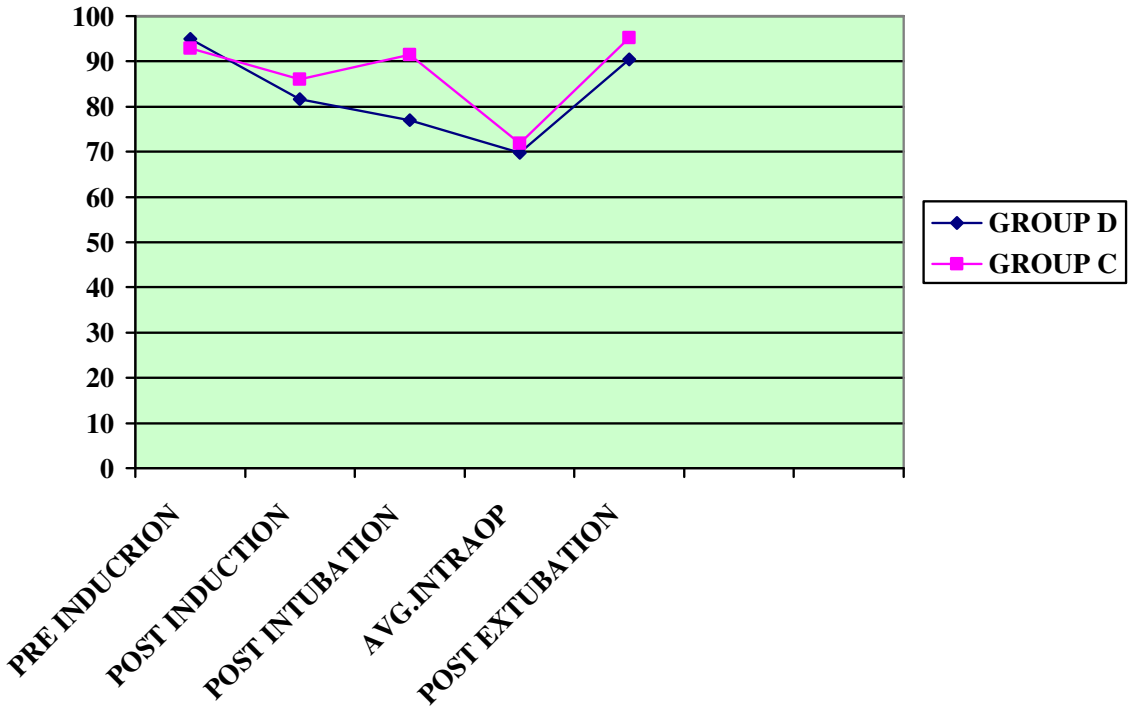
Regarding the systolic blood pressure both groups showed almost equal results in pre induction period. But the a systolic blood pressure after induction, after intubation and during intraoperative period was lower in group D which was statistically significant.

Diastolic Blood Pressure



The diastolic blood pressure was lower in group D after intubation and during the intraoperative period which was statistically significant.

Mean Arterial Pressure



Regarding the mean arterial pressure post intubation & average intra operative values were low in group D which was statistically significant. Other value like pre induction, post induction and post extubation were comparable in both and was not statistically significant.

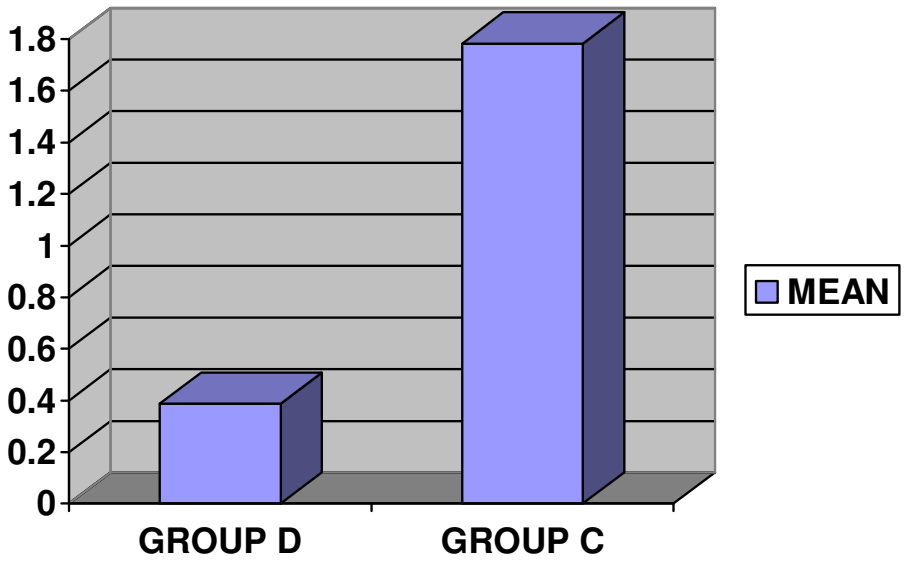
TABLE V

Intraoperative Isoflurane requirement

Student "t"test

	Group D	Group C	p-value
No. of cases	25	25	<0.001*
Mean	0.387	1.783	
S.D	0.102	0.211	
Range	0.2-1.4	1-2.5	

*Statistically Significant



The average intra operative isoflurane requirement was low in group D(0.387) Compared to group C(1.783).This was statistically significant (p- <0.001).

TABLE VI

Intraoperative adverse events

Chi-Square Test

Intraoperative problems		Group D		Group C		p-value
		Nos.	%	Nos.	%	
Hyper tension	YES	0	0	0	0	-
	NO	25	100	25	100	
Hypo tension	YES	0	0	0	0	-
	NO	25	100	25	100	
Arrhythmia	YES	0	0	0	0	-
	NO	25	100	25	100	
Tachycardia	YES	0	0	0	0	-
	NO	25	100	25	100	
Bradycardia	YES	2	8	3	12	1.000
	NO	23	92	22	88	
Ischemia	YES	0	0	0	0	-
	NO	25	100	25	100	

Not statistically significant

Intra operative problems such as hypertension, Hypotension, Arrhythmia, Tachycardia, Ischemia were not seen in any groups. Bradycardia was seen in two cases in group D and three cases in group C. This was not statistically significant (p-1.000)

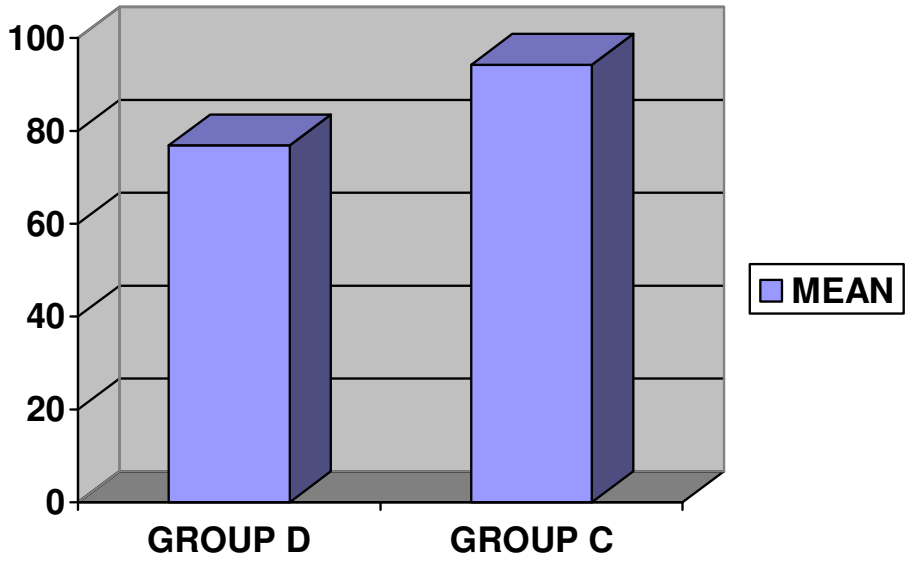
TABLE VII

Effect of Study drug on duration of surgery

Student "t"test

	Group D	Group C	p-value
No. of cases	25	25	0.004*
Mean	76.84	94.16	
S.D	14.174	25.083	
Range	45-102	56-135	

* statistically significant



All patients underwent the same type of surgery. The duration of surgery was less with group D when compared to group C which is statistically significant (P= 0.004).

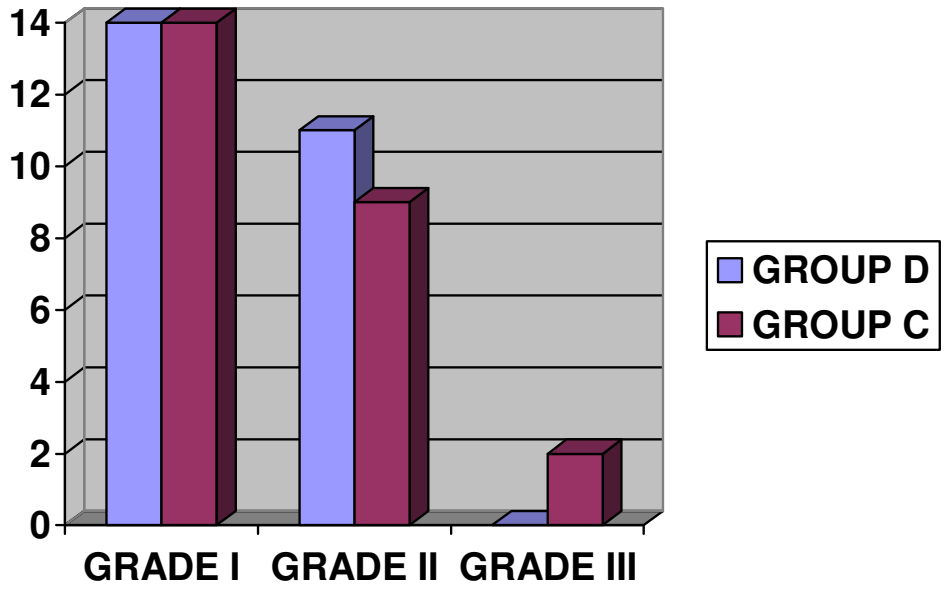
TABLE VIII

Evaluation of the surgical field by surgeon

Chi-Square Test

Fromme Boezzart Scale	Group D	Group C	p-value
No. of cases	25	25	0.333
GRADE I	14	14	
GRADE II	11	09	
GRADE III	00	02	

Not statistically significant



The surgical field grading was almost equal in two groups .Two patients in group C showed Grade III, none in group D. But this was not statistically significant. (P-0.333)

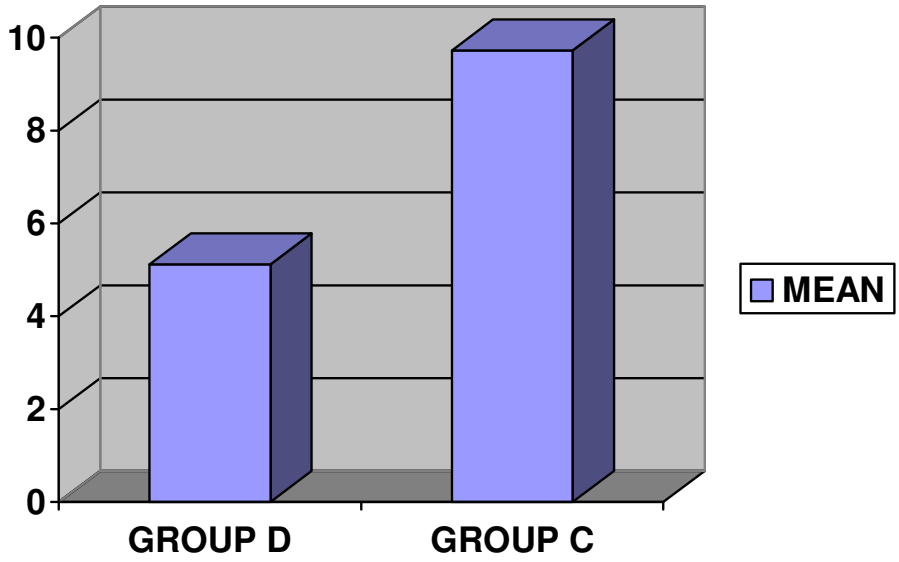
TABLE IX

Awakening Time

Student "t"test

	Group D	Group C	p-value
No. of cases	25	25	<0.001*
Mean	5.12	9.72	
S.D	1.691	1.100	
Range	3-10	8-12	

* statistically significant



The awakening time was low in group D compared to C group which was statistically significant.(p-0.001)

TABLE X

RAMSAY SEDATION SCALE

Group D

Student "t"test

TIME IN MIN	SCORE 2	SCORE 3
15 MIN	15	10
30 MIN	19	06
45 MIN	25	00
60 MIN	25	00

Group C

Student "t"test

TIME IN MIN	SCORE 2	SCORE 3
15 MIN	10	15
30 MIN	18	07
45 MIN	24	01
60 MIN	25	00

There was no significant difference in the sedation score.

CHAPTER IX

DISCUSSION

To achieve controlled hypotension in functional endoscopic sinus surgeries either inhalation technique or intravenous technique are used routinely. If inhalational agents were used to provide hypotensive anesthesia large inspired concentration of the anesthetics was used than that required to produce surgical anesthesia.

By stimulating the pre synaptic alpha 2-adrenoceptors dexmedetomidine decreases the nor epinephrine release and causes fall in blood pressure & heart rate .Because of this property Dexmedetomidine is now a days used as hypotensive agent in endoscopic surgeries. It also has an added advantage of analgesic property thus reducing peri operative analgesic requirement.
Hence this study was designed to evaluate the effect of dexmedetomidine on isoflurane requirement in achieving MAP of 60-70mmHg.

Both the groups were comparable in demographic distribution, resting heart rate and blood pressure.

Even though both the group received same mode of induction, intubation response was less in dexmedetomidine group compared to control. This was comparable with result of **Berrin Isik, et al,**[29]

Intra operative mean arterial pressure was maintained around 60-70mmHg by titrating isoflurane percentage. The group C needed more isoflurane(1.7±0.211) than group D(0.387±0.102).This was statistically significant(p-<0.001). This finding concurred with the results of the study by **Mohammad Maroof, et al**[18]**,** in their study on 'Dexmedetomidine is a Useful Hypotensive Adjunct during Middle Ear Surgery under General Anesthesia'. In their study the mean isoflurane requirement in DEX group was 1.3±0.4 and in saline group was 3.1±0.3.The same result was observed by **Farah Nasreen , et al,** [23].But they used halothane.

The intra operative heart rate was found to be lower in group D. This finding concurred with the results of the study by **Guldem Turan, et al**[20] and **Durmus M, et al**[19] They found that the heart rate was lower in DEX group .This finding also concurred with the results of **Hilal Ayoglu, et al** [21] showed DEX will not cause reflex tachycardia peri operatively. The average intra operative systolic blood pressure , diastolic blood pressure , mean

arterial pressure were significantly lower in Dexmedetomidine group compared to control group.

Regarding the intra operative adverse reaction 2 patients in group D(8%) and 3 patients in group C(12%) developed bradycardia. It was easily reversed by atropine 0.3mg IV single dose in both the groups.

This was not statistically significant (p-1.000). Other intra operative problems like hypertension , arrhythmias ,tachycardia, ischemia were not encountered in either of the groups.

There was statistically significant difference in duration of surgery (p-.004) The mean duration of surgery in group D was (76.84±14.174) In group C was (94.1±25.083).

The evaluation of surgical field was done by a surgeon blinded to the study drugs using Fromme Boezzart scale. All patients in group D belonged to grade 2 and below and in group C to grade 3 and below, which denotes highly acceptable surgical field as far as the surgeon was concerned.

Some studies showed that the need for perioperative analgesic requirement was lower in dex group(**Durmus M, et Al[19], Hilal Ayoglu et al,[21]** and **Iclal Ozdemir Kol,et al, et al[22]**).We haven't analyzed this parameter.

The awakening time in min (time required to tell clearly their name from the end of tracheal extubation) was lower in group D(5.12±1.691) compared to group C(9.72±1.100)It was statistically significance(p-0.001).It concurred with the result of **Mohammad Maroof, et al** [16]. They found that the mean awakening time in dex group was 9.1±2.7 min and in NS group was 12.8±.2min.The intraoperative isoflurane consumption was comparatively more than our study (in group D- 1.3Vs0.38, in group C(3.1 VS 1.7).This may be the reason the awakening time was prolonged in both the group in their study.

Regarding the hemodynamic stability after extubation both group returned to their baseline values.

In our study however, none of the patients in both the groups had nausea and Vomiting in the post operative period. This could be due to prophylactic administration of inj. Ondansetron.

There was no significant difference in the postoperative sedation score in both the groups.

CHAPTER X

SUMMARY

The prospective randomized control study aimed to evaluate the effect of Dexmedetomidine infusion on the requirement of Isoflurane to maintain a mean arterial pressure of 60-70mmHg, Quality of blood less surgical field, duration of surgery and awakening time in patients undergoing Functional Endoscopic Sinus Surgery(FESS) under general anaesthesia

Important conclusions from this study include

1. Group D lowered intra operative blood pressure better than group C.

2. Group D produced lower intraoperative heart rate than group C.

3. Intra operative isoflurane requirement was low in group D compared to group C.

4. Both the group provided better visualization of the surgical field assessed by the surgeon using Fromme Boezzart scale.

5. The duration of surgery was low in Group D.

6. The awakening time after extubation was also low in Group D compared to Group C.

7. There was no difference in postoperative sedation.

CHAPTER XI

CONCLUSION

Dexmedetomidine infusion helps in achieving a targeted reduction in MAP, reduced intraoperative Isoflurane requirement, better blood less field, and faster awakening in patients undergoing Functional Endoscopic Sinus surgery.

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PROFORMA

Prospective randomised control study of Dexmedetomidine for controlled Hypotension in Functional Endoscopic Sinus Surgery (FESS)

Name: _____ Age / Sex: _____ IP No: _____ Weight: _____ Group: _____

Clinical Diagnosis: _____ Surgery: _____

Investigation: Hb _____ B Sugar: _____ B Urea: _____ Sr. Creatinine: ___ Platelets: _____

CI: _____ BT: _____

Monitors: NIBP, ECG, SPO_2

Premedication: _____ Glycopyrrolate 5 mcg / kg
Fentanyl 2 mcg / kg

Study Drug: Time DRUG (BOLUS 1mcg / kg) Infusion 0.5 mcg / kg

_____ _____ Start Time _____ End Time: _____

Induction: Time: _____ Drug: INJ Propofol 2 mg / kg
INJ Vecuronium 0.1 mg / kg

Intubation: Time: _____ ETT Size: _____

Maintance: N_2o ; O_2 + Isoflurane

INTRA OPERATIVE HEMODYNAMIC PARAMETERS

Variables	Pre Induction	Post Induction	Post Intubation	5min	10min	15	20	25	30	35	60	Post Extubation	
1. HR						(Every 5 Min till Extubation)							
2. SBP													
3. DBP													
4. MAP													
5. SPO_2													
6. Isoflurane													
%													

Intra Operative Complications & Management: _____

Surgical Field grading: _____ Duration of Surgery: _____

Reversal Time: _____ Drug: Neostigmine 0.05 mg / kg
Glycopyrrolate mcg / kg

Time of Extubation: _____ Awake Time: _____

Ramsay Sedation Scale: 15min _____ 30 min _____ 45 min _____ 60 min _____

MASTER CHART GROUP - C

SL.NO	NAME	AGE	SEX	WEIGHT	PRE INDUCTION					POST INDUCTION					POST INTUBATION					5 MIN				
					HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	VIOLET	32	F	56	66	106	67	80	NIL	68	96	65	75	1	77	110	70	83	1.4	75	106	55	72	1.8
2	PADMASHINI	48	F	50	82	128	76	93	NIL	84	109	63	78	1	93	124	79	94	1.4	87	122	77	92	1.6
3	SURENDRABABU	19	M	60	74	124	85	98	NIL	81	112	60	77	1	89	140	95	110	1.4	88	118	78	91	1.6
4	THANGAM	55	F	56	90	132	88	103	NIL	82	121	62	82	1	87	119	78	92	1.4	82	112	75	87	1.6
5	PRAKASH	28	M	60	76	124	83	97	NIL	70	107	71	83	1	90	99	66	77	1	80	107	77	87	1.6
6	RAMESH	35	M	60	75	128	83	98	NIL	74	117	79	92	1	94	121	83	96	1.2	79	93	64	74	1.2
7	SUDHAKAR	30	M	55	82	132	88	103	NIL	70	93	67	76	1	96	120	92	101	1.2	89	118	70	86	1.4
8	VEERAMUTHU	23	M	55	67	125	70	88	NIL	65	90	48	62	NIL	90	117	66	83	1	94	119	76	90	1.2
9	ANANDBABU	22	M	55	74	123	79	94	NIL	85	98	61	73	1	99	124	78	93	1.2	97	119	77	91	1.4
10	KAMARAJ	30	M	60	91	122	76	91	NIL	89	126	75	92	1	92	102	69	80	1.2	87	115	81	92	1.6
11	RAMAN	32	M	45	69	124	80	95	NIL	67	127	85	99	1	82	117	86	96	1.6	81	113	84	94	1.8
12	PADMANABHAN	34	M	65	81	128	84	99	NIL	72	121	62	82	1	89	120	82	95	1.4	89	115	71	86	1.6
13	SHANMUGAM	26	M	60	84	118	78	91	NIL	72	104	77	86	1	87	104	68	80	1.2	90	102	60	74	1.4
14	MIRNALINI	27	F	50	72	124	80	95	NIL	90	130	87	101	1	104	129	83	98	1.2	94	101	69	80	1.4
15	YUVARANI	18	F	55	94	108	74	85	NIL	69	110	67	81	1	80	124	87	99	1.2	82	102	68	79	1.4
16	KALAVATHI	29	F	53	98	113	81	92	NIL	72	102	68	79	1	95	108	78	88	1.2	81	96	70	79	1.4
17	ANANDAN	36	M	58	73	119	82	94	NIL	89	122	74	90	1	92	106	65	79	1.2	86	99	64	76	1.4
18	SHAJATHA	29	M	65	80	119	80	93	NIL	97	123	87	99	1	98	150	116	127	1.4	95	139	101	114	2
19	GOWERI	50	F	65	78	139	89	106	NIL	76	142	91	108	1	77	99	68	78	1.2	89	137	94	108	1.8
20	DINESH	24	M	60	93	108	64	79	NIL	68	126	56	79	1	82	108	74	85	1.2	87	113	77	89	1.4
21	SHANKAR	32	M	62	78	130	90	103	NIL	88	132	91	105	1	77	108	74	85	1.4	79	104	61	75	1.6
22	KAUSIKANTH	38	M	60	79	112	64	80	NIL	65	124	74	91	1	83	126	73	91	1.4	86	125	73	90	1.6
23	PONGUNDRAN	26	M	65	75	122	74	90	NIL	70	100	67	78	1	86	122	75	91	1.2	78	113	69	84	1.4
24	PREMA	53	F	55	68	105	75	85	NIL	66	122	84	97	1	75	119	81	94	1.2	68	128	87	101	1.6
25	RADHA	18	F	50	82	115	78	90	NIL	83	118	69	85	1	87	123	74	90	1.4	90	129	78	95	1.8

SL.NO	10 MIN					15 MIN					20 MIN					25 MIN					30 MIN					35 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	79	86	58	67	1.4	79	87	58	68	1.4	82	90	58	69	1.4	89	97	62	74	1.6	90	89	60	70	1.6	81	90	58	69	1.6
2	75	112	63	79	1.8	73	101	62	75	2	69	89	61	70	2	66	88	59	69	1.8	71	89	57	68	1.8	78	86	58	67	1.6
3	77	106	61	76	1.8	79	99	57	71	1.8	87	101	63	76	2	87	89	61	70	2	88	89	59	69	2	86	89	57	68	2
4	86	97	69	78	1.8	78	94	66	75	2	77	94	61	72	2	79	90	58	69	2	84	89	57	68	2	85	89	56	67	1.8
5	74	96	62	73	1.6	76	94	66	75	1.8	70	96	68	77	2	72	92	60	71	2	69	90	59	69	2	70	93	59	70	2
6	89	100	72	81	1.4	81	98	73	81	1.6	83	97	73	81	1.8	89	93	70	78	1.8	81	95	70	78	2	75	106	70	82	2.5
7	76	101	68	79	1.6	65	92	52	65	1.4	69	108	68	81	1.6	64	92	56	68	1.6	60	93	56	68	1.6	62	93	55	68	1.6
8	82	102	61	75	1.4	63	102	60	74	1.6	74	94	55	68	1.6	67	105	56	72	1.8	67	103	56	72	2	76	103	55	71	2
9	89	91	63	72	1.6	76	91	63	72	1.8	71	91	63	72	2	72	91	61	71	2	74	91	60	70	2	75	91	59	70	2
10	83	113	78	90	1.8	85	113	72	86	2	71	112	64	80	2.5	76	89	61	70	2	79	89	59	69	2	73	85	57	66	2
11	80	113	82	92	2	71	103	73	83	2.5	62	94	67	76	2.5	57	87	62	70	2	56	86	56	66	2	55	85	55	65	2
12	78	90	59	69	1.6	79	89	59	69	1.6	78	89	57	68	1.6	76	88	60	69	1.6	81	87	59	68	1.6	82	89	60	70	1.6
13	78	89	59	69	1.4	75	87	58	68	1.4	69	91	59	70	1.6	78	90	59	69	1.6	79	89	56	67	1.6	78	89	57	68	1.6
14	86	98	67	77	1.6	93	85	53	64	1.4	93	109	66	80	1.6	93	109	66	80	1.8	85	92	53	66	1.8	78	92	57	69	1.8
15	83	99	68	78	1.8	88	93	62	72	1.8	80	94	67	76	2	86	89	64	72	2	82	90	60	70	2	84	87	58	68	2
16	82	97	70	79	1.6	83	97	70	79	1.8	83	95	70	78	2	86	84	51	62	1.8	84	89	52	64	1.6	87	89	54	66	1.6
17	68	90	58	69	1.4	72	89	57	68	1.4	79	88	58	68	1.2	82	91	62	72	1.4	65	90	58	69	1.4	69	89	57	68	1.4
18	95	111	67	82	2	89	99	56	70	2	88	96	57	70	2	89	94	56	69	2	79	88	53	65	2	82	87	53	64	1.8
19	94	134	89	104	2	93	124	84	97	2	92	99	62	74	2.5	93	97	60	72	2.5	90	93	62	72	2.5	90	89	61	70	2.5
20	83	102	68	79	1.6	85	111	70	84	1.8	81	105	70	82	2	82	85	57	66	2	68	88	58	68	2	69	90	60	70	2
21	83	90	61	71	1.6	85	101	66	78	1.8	82	97	60	72	2	78	90	58	69	2	79	88	58	68	2	81	90	58	69	2
22	88	112	67	82	2	74	95	64	74	2	78	91	59	70	2	69	89	57	68	2	71	89	59	69	2	75	86	58	67	1.8
23	83	106	69	81	1.6	87	95	61	72	1.6	77	97	62	74	1.8	69	97	62	74	2	65	89	58	68	2	69	88	59	69	2
24	66	131	85	100	1.8	68	118	76	90	2	70	114	64	81	2.5	73	91	59	70	2	71	89	59	69	2	72	89	57	68	2
25	89	118	75	89	1.8	87	109	69	82	2	88	105	67	80	2.5	89	96	62	73	2.5	86	88	60	69	2.5	84	88	56	67	2

SL.NO	40 MIN					45 MIN					50 MIN					55 MIN					60 MIN					65 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	85	86	59	68	1.6	85	86	57	67	1.6	83	88	59	69	1.4	80	98	58	71	1.6	85	93	62	72	1.8	83	88	58	68	1.8
2	72	86	56	66	1.6	73	89	61	70	1.6	76	90	62	71	1.8	79	88	59	69	1.8	74	88	57	67	1.8	74	89	57	68	1.8
3	78	90	58	69	2	76	87	55	66	2	82	89	57	68	2	75	90	56	67	2	72	109	71	84	NIL	71	110	65	80	NIL
4	75	88	58	68	1.8	78	91	59	70	1.8	74	92	60	71	2	79	89	56	67	2	82	89	59	69	2	87	90	58	69	2
5	82	90	58	69	2	75	87	56	66	2	75	87	58	68	2	71	87	57	67	1.8	67	87	59	68	1.8	69	89	59	69	1.8
6	80	90	62	71	2.5	85	89	61	70	2.5	89	88	60	69	2.5	88	88	59	69	2	87	88	58	68	2	89	90	60	70	2
7	61	93	55	68	1.6	59	92	54	67	1.4	59	98	54	69	1.4	57	90	55	67	1.4	53	90	55	67	1.4	55	91	60	70	1.4
8	79	100	55	70	2	82	96	56	69	2	81	97	56	70	2	83	98	58	71	2	88	94	58	70	2	88	94	57	69	2
9	72	90	58	69	2	70	88	57	67	1.8	66	87	58	68	1.8	64	87	57	67	1.8	60	87	56	66	1.6	61	86	55	65	1.6
10	76	88	59	69	2	75	89	57	68	2	73	89	56	67	1.8	72	89	57	68	1.6	71	91	62	72	1.8	72	89	59	69	1.8
11	54	85	55	65	1.8	52	85	55	65	1.8	65	89	70	76	2	65	89	68	75	2	68	86	64	71	2	69	85	62	70	2
12	84	92	62	72	1.8	85	90	60	70	1.8	78	90	58	69	1.8	76	88	57	67	1.8	71	89	55	66	1.6	76	88	59	69	1.6
13	69	90	58	69	1.6	67	90	60	70	1.8	71	89	59	69	1.8	73	89	58	68	1.8	74	85	57	66	1.6	79	85	55	65	1.4
14	76	92	55	67	1.8	77	92	55	67	1.8	76	92	56	68	1.8	78	92	57	69	1.8	74	91	57	68	1.8	72	100	72	81	NIL
15	88	86	55	65	1.8	89	93	62	72	2	89	92	60	71	2	89	90	59	69	2	86	90	59	69	2	89	87	57	67	2
16	80	87	55	66	1.6	78	88	55	66	1.6	76	90	56	67	1.6	71	89	59	69	1.6	69	89	56	67	1.6	70	96	56	69	NIL
17	78	86	56	66	1.2	76	89	61	70	1.4	69	91	59	70	1.6	70	87	57	67	1.6	67	89	57	68	1.6	69	90	58	69	1.6
18	78	86	53	64	1.8	79	84	52	63	1.6	79	85	51	62	1.4	76	83	50	61	1.2	70	83	50	61	1	72	84	51	62	1
19	85	88	61	70	2.5	85	90	60	70	2.5	83	88	59	69	2	80	88	58	68	2	85	102	67	79	2.5	83	90	59	69	2
20	67	90	58	69	2	80	88	57	67	2	78	87	56	66	1.8	83	88	59	69	1.8	85	91	59	70	2	81	88	60	69	2
21	82	88	58	68	1.8	86	91	59	70	1.8	84	89	59	69	1.8	76	88	56	67	1.8	77	89	59	69	1.8	74	88	57	67	1.6
22	72	91	59	70	2	70	88	58	68	2	81	88	57	67	2	78	90	58	69	2	82	91	60	70	NIL	87	96	61	73	NIL
23	70	86	58	67	2	67	90	58	69	2	65	89	56	67	1.8	78	90	58	69	1.6	81	88	57	67	1.6	81	91	62	72	1.8
24	72	86	57	67	1.8	68	90	57	68	1.8	67	88	59	69	1.6	69	91	60	70	1.8	75	90	59	69	1.8	73	90	60	70	NIL
25	85	89	59	69	2	85	91	62	72	2.5	83	90	59	69	2	80	89	57	68	2	85	90	55	67	2	83	90	58	69	2

SL.NO	70 MIN					75 MIN					80 MIN					85 MIN					90 MIN					95 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	71	90	58	69	1.8	69	88	57	67	1.8	75	86	56	66	1.4	78	89	57	68	1.4	82	90	58	69	1.4	84	94	58	70	NIL
2	78	88	57	67	1.6	68	89	61	70	NIL	69	91	62	72	NIL	76	94	62	73	NIL									
3																													
4	85	87	59	68	2	85	89	57	68	2	76	86	57	67	1.8	78	85	57	66	1.8	76	88	56	67	1.6	70	89	59	69	1.6
5	69	85	59	68	1.8	66	88	59	69	1.8	66	90	59	69	1.8	65	88	59	69	1.8	66	90	59	69	1.8	65	86	58	67	1.6
6	85	89	61	70	2	84	89	60	70	2	93	90	61	71	2	94	89	60	70	2	89	90	60	70	2	75	107	71	83	NIL
7	53	92	61	71	1.6	53	91	60	70	1.6	53	90	60	70	1.6	52	90	60	70	1.6	75	123	90	101	NIL	89	134	90	105	NIL
8	76	93	56	68	1.8	73	105	57	73	2	75	91	59	70	2	77	92	58	69	2	78	89	59	69	1.8	83	106	64	78	NIL
9	61	85	55	65	1.6	59	85	55	65	1.4	57	84	55	65	1.4	55	85	55	65	1.2	56	88	56	67	1.2	55	88	57	67	1.2
10	76	89	58	68	1.8	78	88	57	67	1.6	87	89	61	70	1.8	79	88	59	69	1.6	76	88	61	70	NIL	77	88	58	68	NIL
11	68	85	62	70	2	70	85	61	69	2	71	84	61	69	2	71	83	62	69	2	70	83	62	69	2	68	82	60	67	1.8
12	76	89	61	70	1.6	72	88	60	69	NIL	75	95	61	72	NIL	87	115	66	82	NIL									
13	77	87	62	70	1.6	73	89	62	71	1.8	78	89	59	69	1.8	67	89	57	68	1.8	65	89	61	70	2	69	90	60	70	2
14	72	100	72	81	NIL	74	104	73	83	NIL	73	110	79	89	NIL														
15	89	86	56	66	1.8	86	83	53	63	1.6	82	86	68	74	1.8	83	91	64	73	2	88	89	61	70	2	88	89	59	69	2
16	72	98	60	73	NIL	85	98	62	74	NIL																			
17	65	89	55	66	1.4	72	89	56	67	1.4	73	88	58	68	1.4	65	89	59	69	1.4	64	89	58	68	1.4	62	88	56	67	1.4
18	70	86	51	63	0.8	67	87	51	63	0.8	67	87	53	64	0.8	65	88	52	64	0.6	63	97	60	72	0.8	65	96	60	72	0.8
19	71	95	64	74	NIL	69	93	62	72	NIL	75	121	82	95	NIL														
20	78	89	57	68	1.8	76	89	59	69	1.8	76	88	60	69	1.8	78	89	61	70	1.8	70	90	61	71	NIL	69	103	74	84	NIL
21	68	90	58	69	1.6	67	88	60	69	1.6	69	89	61	70	1.8	66	89	59	69	1.8	64	89	58	68	1.6	67	88	60	69	1.6
22	82	97	62	74	NIL	78	106	74	85	NIL																			
23	79	89	59	69	1.8	77	90	57	68	1.8	78	91	58	69	1.8	77	90	59	69	NIL	79	92	63	73	NIL	86	109	65	80	NIL
24	68	96	69	78	NIL	65	109	68	82	NIL	67	125	87	100	NIL														
25	87	88	57	67	2	85	89	58	68	2	82	90	57	68	2	80	89	59	69	NIL	78	93	60	71	NIL	77	112	67	82	NIL

SL.NO	100 MIN					105 MIN					110 MIN					115 MIN					120 MIN					125 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	74	96	58	71	NIL	76	94	64	74	NIL	70	96	66	76	NIL															
2																														
3																														
4	78	90	60	70	1.8	73	90	58	69	1.8	71	89	57	68	NIL	73	91	62	72	NIL	87	97	62	74	NIL					
5	66	86	58	67	1.6	65	90	58	69	1.6	65	87	57	67	1.4	68	89	57	68	1.4	65	84	56	65	1.2	69	86	58	67	NIL
6	75	132	92	105	NIL	75	123	78	93	NIL	78	116	77	90	NIL															
7																														
8	73	121	69	86	NIL	85	119	80	93	NIL																				
9	55	88	57	67	1.2	53	89	57	68	1.2	56	87	57	67	1	54	86	57	67	1	51	87	58	68	NIL	66	113	79	90	NIL
10	78	91	62	72	NIL																									
11	66	82	60	67	1.8	65	85	61	69	1.8	64	86	62	70	1.8	60	88	60	69	1.8	54	96	71	79	NIL	54	102	73	83	NIL
12																														
13	76	88	57	67	1.8	76	89	59	69	1.8	78	88	56	67	NIL	76	88	60	69	NIL	76	90	60	70	NIL	68	97	65	76	NIL
14																														
15	87	90	59	69	2	87	90	57	68	2	89	93	60	71	2	91	87	54	65	2	92	86	53	64	1.8	89	87	54	65	1.8
16																														
17	58	90	57	68	1.4	50	90	58	69	1.4	78	90	60	70	1.6	82	89	61	70	1.6	79	90	58	69	1.4	81	89	58	68	NIL
18	61	95	59	71	1	68	96	58	71	1.2	65	94	58	70	1.2	67	92	58	69	1.2	63	91	56	68	1.2	63	88	55	66	1
19																														
20	79	133	84	100	NIL																									
21	71	95	62	73	NIL	72	91	60	70	NIL	75	98	66	77	NIL	78	100	60	73	NIL										
22																														
23	85	122	89	100	NIL																									
24																														
25	78	128	94	105	NIL																									

SL.NO	130 MIN					135 MIN					140 MIN					145 MIN					150 MIN					AFTER EXTUBATION			
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP
1																										62	118	78	91
2																										76	118	70	86
3																										74	118	72	87
4																										92	108	74	85
5	68	88	60	69	NIL	69	87	61	70	NIL	68	89	61	70	NIL											83	110	77	88
6																										68	130	80	97
7																										69	103	68	80
8																										65	124	66	85
9	76	128	98	108	NIL																					87	125	81	96
10																										68	118	70	86
11	54	106	75	85	NIL	52	108	75	86	NIL																64	141	99	113
12																										81	127	83	98
13																										92	115	74	88
14																										89	124	83	97
15	80	85	54	64	NIL	64	97	68	78	NIL	64	97	68	78	NIL											84	128	76	93
16																										86	124	88	100
17	78	92	63	73	NIL	84	123	84	97	NIL																72	120	84	96
18	68	91	56	68	1	72	91	53	66	1	74	91	56	68	NIL	75	101	74	83	NIL	103	142	108	119	NIL	113	149	110	123
19																										74	124	86	99
20																										72	120	81	94
21																										83	132	84	100
22																										72	136	89	105
23																										89	136	83	101
24																										89	128	87	101
25																										72	124	76	92

SL.NO	INTRAOP EVENTS						DURATION OF SURGERY	FROMME BOEZART SCALE
	HYPER TENSION	HYPO TENSION	ARRYTHMIAS	ISCHEMIA	TACHYCARDIA	BRADYCARDIA		
1	N	N	N	N	N	N	94	1
2	N	N	N	N	N	N	85	2
3	N	N	N	N	N	N	56	1
4	N	N	N	N	N	N	112	2
5	N	N	N	N	N	N	130	1
6	N	N	N	N	N	N	95	1
7	N	N	N	N	N	N	72	1
8	N	N	N	N	N	N	75	2
9	N	N	N	N	N	Y	120	2
10	N	N	N	N	N	N	102	2
11	N	N	N	N	N	Y	120	1
12	N	N	N	N	N	N	82	1
13	N	N	N	N	N	N	110	1
14	N	N	N	N	N	N	65	1
15	N	N	N	N	N	N	133	3
16	N	N	N	N	N	N	62	2
17	N	N	N	N	N	Y	130	2
18	N	N	N	N	N	N	135	1
19	N	N	N	N	N	N	60	1
20	N	N	N	N	N	N	92	1
21	N	N	N	N	N	N	109	1
22	N	N	N	N	N	N	66	3
23	N	N	N	N	N	N	96	2
24	N	N	N	N	N	N	68	1
25	N	N	N	N	N	N	85	2

SL.NO	AWAKENING TIME	RAMSAY SEDATION SCALE			
		15	30	45	60
1	10	3	3	2	2
2	11	3	3	2	2
3	10	3	2	2	2
4	9	2	2	2	2
5	10	3	3	2	2
6	10	3	2	2	2
7	8	2	2	2	2
8	9	3	2	2	2
9	8	2	2	2	2
10	10	3	2	2	2
11	10	3	2	2	2
12	11	3	3	2	2
13	9	2	2	2	2
14	9	2	2	2	2
15	10	3	2	2	2
16	8	2	2	2	2
17	11	3	3	2	2
18	11	3	2	2	2
19	12	3	3	3	2
20	9	2	2	2	2
21	10	3	2	2	2
22	9	2	2	2	2
23	8	2	2	3	2
24	10	2	2	2	2
25	11	3	3	3	2

MASTER CHART GROUP - D

SL.NO	NAME	AGE	SEX	WEIGHT	PRE INDUCTION					POST INDUCTION					POST INTUBATION					5 MIN				
					HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	VASUKI	30	F	50	74	130	74	93	NIL	67	96	69	78	1	72	90	60	70	0.8	65	89	58	68	0.6
2	MOHAN	42	M	62	80	120	88	99	NIL	75	100	60	73	1	66	92	60	71	0.8	65	92	59	70	0.8
3	MUTHU	22	M	60	85	132	89	103	NIL	81	126	82	97	1	84	128	79	95	1.2	73	102	60	74	1.2
4	LALITHA	38	F	63	86	112	81	91	NIL	82	104	72	83	1	86	100	65	77	1	73	90	61	71	0.6
5	MANIVEL	26	M	56	70	130	90	103	NIL	64	106	54	71	1	76	112	63	79	1.2	72	100	63	75	1.4
6	ARUN	26	M	66	68	135	81	99	NIL	63	125	71	89	1	81	134	84	101	1.2	85	100	63	75	1.2
7	DRAVIDASELVAN	42	M	65	81	140	82	101	NIL	76	93	64	74	1	84	112	79	90	1.2	82	99	65	76	1.2
8	SANGETHA	28	F	52	76	121	79	93	NIL	65	98	70	79	1	76	92	60	71	1	64	90	58	69	0.8
9	KAMARAJ	30	M	58	81	108	75	86	NIL	70	100	66	77	1	65	90	60	70	0.8	63	92	64	73	0.8
10	KARTHIKEYAN	50	M	65	72	118	72	87	NIL	63	106	67	80	1	76	98	60	73	1	67	96	55	69	0.6
11	DEVI	24	F	45	70	124	89	101	NIL	83	121	86	98	1	74	104	69	81	1.2	72	95	60	72	1.2
12	ASHOK	36	M	55	75	138	94	109	NIL	69	118	87	97	1	77	106	76	86	1.2	65	96	62	73	1.2
13	KUMAR	36	M	70	76	130	87	101	NIL	66	122	73	89	1	71	96	68	77	1.2	64	95	57	70	1
14	SASIKUMAR	35	M	65	85	120	82	95	NIL	71	104	71	82	1	85	90	60	70	0.8	68	90	59	69	0.8
15	VATCHALA	32	F	65	82	124	87	99	NIL	85	96	70	79	1	72	90	59	69	0.8	73	90	58	69	0.6
16	UTHARA	19	F	50	84	122	81	95	NIL	69	114	80	91	1	68	96	61	73	1.2	65	90	59	69	1
17	SUDHAKAR	30	M	60	69	116	80	92	NIL	80	96	61	73	1	76	121	78	92	1.4	75	112	60	77	1.4
18	JAGADESHVARAN	34	M	62	79	109	73	85	NIL	68	106	67	80	1	71	113	69	84	1.2	67	90	62	71	1.2
19	DURAIRAJ	40	M	65	77	122	88	99	NIL	69	99	63	75	1	72	90	60	70	0.8	62	89	56	67	0.6
20	LAKSHMIKANTH	38	M	65	69	113	69	84	NIL	72	106	69	81	1	74	94	63	73	1	72	90	59	69	0.8
21	GANESAN	48	M	60	75	114	80	91	NIL	70	90	68	75	1	73	90	59	69	0.8	64	90	59	69	0.6
22	KARTHIK	18	M	60	69	119	75	90	NIL	65	119	74	89	1	74	90	60	70	0.8	66	91	59	70	0.8
23	PREMA	53	F	50	70	122	78	93	NIL	68	99	62	74	1	71	95	60	72	1	67	90	60	70	0.6
24	SUJATHA	35	F	52	72	120	81	94	NIL	82	96	63	74	1	82	93	60	71	1	61	89	56	67	0.8
25	SARAVANAN	18	M	55	73	118	79	92	NIL	69	109	67	81	1	72	105	54	71	1	66	97	60	72	1.2

SL.NO	10 MIN					15 MIN					20 MIN					25 MIN					30 MIN					35 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	55	90	59	69	0.6	55	99	62	74	0.8	55	90	61	71	0.8	56	86	58	67	0.8	60	86	51	63	0.4	54	84	51	62	0.2
2	55	89	58	68	0.6	55	88	61	70	0.6	55	89	56	67	0.4	56	86	58	67	0.2	60	86	51	63	NIL	54	84	51	62	NIL
3	65	92	58	69	1	58	89	55	66	0.6	56	88	56	67	0.6	58	89	2	31	0.6	60	86	57	67	0.4	59	84	54	64	0.2
4	67	89	59	69	0.6	66	89	58	68	0.4	62	88	58	68	0.4	56	96	58	71	0.6	57	86	55	65	0.4	58	88	54	65	0.2
5	55	90	61	71	1.4	55	89	62	71	1	55	89	61	70	0.8	56	96	58	71	0.8	60	86	51	63	0.4	54	84	51	62	0.2
6	55	95	60	72	1.2	55	90	60	70	1	55	89	61	70	0.8	56	86	58	67	0.6	60	86	51	63	0.4	54	87	54	65	0.2
7	65	90	58	69	0.6	63	90	58	69	0.4	63	81	52	62	0.2	59	83	53	63	0.2	59	86	53	64	0.2	57	89	55	66	NIL
8	58	89	60	70	0.6	55	90	56	67	0.4	55	88	52	64	0.2	56	88	53	65	NIL	54	86	52	63	NIL	54	89	56	67	NIL
9	62	90	59	69	0.6	59	89	60	70	0.6	62	90	59	69	0.6	56	96	58	71	0.8	60	89	59	69	0.6	56	89	57	68	0.4
10	61	89	58	68	0.6	57	89	59	69	0.6	55	88	57	67	0.4	56	86	52	63	NIL	60	86	55	65	NIL	58	89	58	68	NIL
11	68	84	57	66	0.6	72	86	56	66	0.4	69	89	60	70	0.4	66	86	60	69	0.4	69	89	61	70	0.4	66	89	59	69	0.4
12	55	90	60	70	1.2	55	89	60	70	1	55	88	60	69	0.8	58	86	57	67	0.6	60	86	56	66	0.4	59	87	58	68	0.4
13	62	90	59	69	0.6	60	89	58	68	0.6	62	91	57	68	0.4	60	93	58	70	0.4	56	88	60	69	0.4	54	80	54	63	NIL
14	61	89	60	70	0.6	56	90	59	69	0.6	55	89	61	70	0.6	56	92	58	69	0.6	60	86	59	68	0.4	56	84	53	63	0.2
15	65	84	57	66	0.4	59	81	56	64	0.2	57	86	54	65	0.2	55	87	58	68	0.2	53	88	59	69	0.2	54	87	59	68	0.2
16	58	89	59	69	0.8	55	86	58	67	0.6	60	90	61	71	0.8	56	86	58	67	0.6	58	86	58	67	0.4	54	84	56	65	0.2
17	59	90	59	69	0.8	55	88	57	67	0.6	55	89	60	70	0.6	56	88	58	68	0.4	60	87	56	66	0.4	54	89	55	66	0.2
18	63	91	58	69	1	61	86	53	64	0.8	55	89	60	70	0.8	56	86	56	66	0.8	58	86	58	67	0.6	54	89	55	66	0.4
19	55	88	59	69	0.6	55	87	60	69	0.4	55	86	53	64	0.2	56	92	58	69	0.2	57	90	56	67	0.2	61	89	60	70	0.4
20	66	90	58	69	0.6	65	89	58	68	0.4	63	85	56	66	0.4	59	83	53	63	0.2	59	86	53	64	0.2	57	89	55	66	NIL
21	60	89	59	69	0.6	61	86	58	67	0.4	57	90	56	67	0.2	56	90	58	69	0.2	58	90	57	68	0.2	58	89	58	68	0.2
22	60	90	58	69	0.6	60	89	58	68	0.6	63	82	52	62	0.2	59	82	52	62	NIL	59	85	52	63	NIL	58	86	53	64	NIL
23	62	89	63	72	0.8	58	86	61	69	0.8	55	88	59	69	0.6	56	89	58	68	0.6	60	86	55	65	0.4	54	86	55	65	0.4
24	55	88	58	68	0.6	61	90	63	72	0.8	57	89	58	68	0.6	56	87	58	68	0.4	59	88	55	66	0.4	57	89	56	67	0.4
25	67	90	59	69	0.8	66	90	60	70	0.8	64	90	62	71	1	68	89	55	66	0.8	63	85	53	64	0.6	66	88	52	64	0.4

SL.NO	40 MIN					45 MIN					50 MIN					55 MIN					60 MIN					65 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	51	92	62	72	0.4	51	93	57	69	0.4	51	99	55	70	0.4	52	92	57	69	0.2	51	101	55	70	0.2	52	93	57	69	0.2
2	51	90	62	71	NIL	56	100	57	71	0.2	55	99	55	70	0.2	53	96	56	69	0.2	57	97	55	69	NIL	60	98	59	72	NIL
3	61	92	58	69	0.2	62	93	55	68	0.2	59	99	55	70	0.4	54	90	58	69	0.4	56	89	57	68	0.2	56	90	57	68	0.2
4	55	90	57	68	0.2	53	88	59	69	0.2	51	89	55	66	0.2	52	86	55	65	0.2	54	90	57	68	0.2	56	91	59	70	NIL
5	51	92	62	72	0.2	51	121	57	78	0.4	51	99	55	70	0.4	52	99	55	70	0.2	51	101	55	70	0.2	52	100	57	71	0.4
6	55	89	58	68	0.2	56	90	57	68	0.2	53	89	56	67	0.2	57	88	59	69	0.2	56	102	60	74	NIL	63	100	62	75	NIL
7	57	100	61	74	0.2	57	92	60	71	0.2	57	96	63	74	0.4	57	90	59	69	0.4	56	97	63	74	0.6	55	90	58	69	0.4
8	56	91	60	70	0.2	54	90	57	68	0.2	55	89	55	66	0.2	58	90	59	69	0.2	57	90	58	69	0.2	56	90	60	70	0.2
9	57	88	58	68	0.4	54	89	57	68	0.4	57	92	56	68	0.4	58	89	54	66	0.2	57	90	59	69	0.2	59	89	57	68	0.2
10	51	92	62	72	0.2	56	90	57	68	0.2	55	92	58	69	0.2	56	91	55	67	0.2	56	89	56	67	0.2	60	89	58	68	0.2
11	68	89	63	72	0.6	72	89	60	70	0.6	71	91	60	70	0.6	70	89	58	68	0.4	66	86	59	68	0.4	63	91	60	70	0.4
12	56	90	55	67	0.2	58	89	60	70	0.2	59	90	56	67	0.2	57	89	59	69	0.2	56	86	58	67	0.2	58	92	60	71	NIL
13	52	81	55	64	NIL	53	87	55	66	NIL	53	85	55	65	NIL	54	87	55	66	NIL	56	93	62	72	0.2	55	90	60	70	0.2
14	55	90	57	68	0.2	54	91	57	68	0.2	56	96	59	71	0.4	61	92	55	67	0.4	63	89	57	68	0.2	62	90	58	69	NIL
15	56	96	64	75	0.4	53	96	63	74	0.6	57	87	62	70	0.6	54	87	62	70	0.6	55	87	60	69	0.6	59	88	64	72	NIL
16	57	92	62	72	0.4	62	89	57	68	0.4	60	89	58	68	0.4	55	90	59	69	0.2	56	91	57	68	0.2	59	90	58	69	NIL
17	61	90	56	67	0.2	56	89	57	68	0.2	60	90	59	69	0.2	62	90	60	70	0.2	63	91	62	72	NIL	61	90	60	70	NIL
18	58	88	59	69	0.4	56	89	57	68	0.4	59	90	55	67	0.4	56	86	54	65	0.2	54	87	55	66	NIL	59	87	56	66	NIL
19	56	90	57	68	0.2	60	89	59	69	0.2	58	90	55	67	0.2	54	89	60	70	0.2	57	90	58	69	0.2	58	89	59	69	NIL
20	57	96	61	73	0.2	57	89	62	71	0.4	57	89	59	69	0.4	57	87	60	69	0.4	56	95	61	72	0.6	55	90	58	69	0.6
21	55	92	63	73	0.4	56	89	57	68	0.4	62	90	55	67	0.2	57	88	55	66	0.2	59	89	55	66	0.2	56	90	62	71	0.4
22	56	89	55	66	NIL	56	89	57	68	NIL	57	91	60	70	NIL	61	101	69	80	NIL	62	97	64	75	NIL					
23	58	88	59	69	0.4	60	90	57	68	NIL	62	97	60	72	NIL	62	108	62	77	NIL										
24	58	90	56	67	0.4	56	90	57	68	0.4	54	86	55	65	0.2	56	88	54	65	NIL	57	90	60	70	0.2	55	89	58	68	0.2
25	61	91	57	68	0.4	64	92	64	73	0.6	68	90	58	69	0.4	65	91	57	68	0.4	65	88	59	69	0.4	64	87	58	68	0.4

SL.NO	70 MIN					75 MIN					80 MIN					85 MIN					90 MIN					95 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	52	92	58	69	0.2	52	91	59	70	0.2	56	100	59	73	NIL	58	98	62	74	NIL	58	98	63	75	NIL	60	100	63	75	NIL
2	59	99	62	74	NIL																									
3	52	89	58	68	0.2	58	90	59	69	NIL	65	96	64	75	NIL	65	97	63	74	NIL										
4	56	96	60	72	NIL	62	100	68	79	NIL																				
5	52	89	58	68	0.2	52	91	59	70	0.2	52	90	60	70	0.4	54	100	64	76	NIL	52	106	75	85	NIL	52	119	86	97	
6	65	104	62	76	NIL																									
7	58	89	60	70	0.4	56	82	50	61	NIL	54	84	52	63	NIL	58	86	54	65	0.2	67	114	83	93	NIL	69	118	76	90	NIL
8	55	89	61	70	0.2	57	92	59	70	0.4	59	89	55	66	0.4	56	88	54	65	0.2	54	89	59	69	0.2	56	90	59	69	NIL
9	60	90	62	71	0.4	56	96	55	69	0.4	59	94	58	70	0.4	61	94	56	69	0.4	60	92	59	70	NIL	63	93	61	72	NIL
10	64	90	60	70	NIL	61	89	58	68	NIL	62	96	55	69	NIL	65	106	65	79	NIL										
11	66	90	59	69	0.4	67	91	58	69	0.4	69	86	56	66	0.2	65	95	60	72	NIL	67	92	62	72	NIL	63	92	64	73	NIL
12	57	95	59	71	NIL	58	96	61	73	NIL	61	96	64	75	NIL															
13	54	90	59	69	0.2	57	90	61	71	NIL	55	88	61	70	NIL	59	89	62	71	NIL	65	115	84	94	NIL					
14	60	91	58	69	NIL	62	96	63	74	NIL	68	126	83	97	NIL															
15	68	124	88	100	NIL																									
16	60	89	61	70	NIL	59	90	62	71	NIL	60	90	63	72	NIL	62	112	76	88	NIL										
17	62	90	61	71	NIL	64	117	75	89	NIL																				
18	59	89	61	70	0.2	60	90	62	71	NIL	64	90	59	69	NIL	67	99	65	76	NIL	68	121	67	85	NIL					
19	58	92	61	71	NIL	65	96	68	77	NIL	68	106	66	79	NIL															
20	58	89	60	70	0.6	56	82	50	61	0.2	54	84	52	63	0.2	58	84	52	63	NIL	67	104	63	77	NIL	69	108	76	87	NIL
21	56	88	56	67	0.2	59	89	58	68	0.2	60	90	57	68	0.2	62	89	57	68	0.2	60	88	59	69	NIL	63	90	64	73	NIL
22																														
23																														
24	56	89	57	68	0.2	53	86	59	68	0.2	57	87	58	68	NIL	57	87	62	70	NIL	56	90	64	73	NIL	60	92	61	71	NIL
25	62	85	58	67	0.2	60	84	56	65	0.2	59	88	59	69	NIL	63	95	61	72	NIL	65	98	61	73	NIL					

SL.NO	100 MIN					105 MIN					110 MIN					115 MIN					120 MIN					125 MIN				
	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %	HR	SBP	DBP	MAP	ISO %
1	74	96	58	71	NIL	76	94	64	74	NIL	70	96	66	76	NIL															
2																														
3																														
4	78	90	60	70	1.8	73	90	58	69	1.8	71	89	57	68	NIL	73	91	62	72	NIL	87	97	62	74	NIL					
5	66	86	58	67	1.6	65	90	58	69	1.6	65	87	57	67	1.4	68	89	57	68	1.4	65	84	56	65	1.2	69	86	58	67	NIL
6	75	132	92	105	NIL	75	123	78	93	NIL	78	116	77	90	NIL															
7																														
8	73	121	69	86	NIL	85	119	80	93	NIL																				
9	55	88	57	67	1.2	53	89	57	68	1.2	56	87	57	67	1	54	86	57	67	1	51	87	58	68	NIL	66	113	79	90	NIL
10	78	91	62	72	NIL																									
11	66	82	60	67	1.8	65	85	61	69	1.8	64	86	62	70	1.8	60	88	60	69	1.8	54	96	71	79	NIL	54	102	73	83	NIL
12																														
13	76	88	57	67	1.8	76	89	59	69	1.8	78	88	56	67	NIL	76	88	60	69	NIL	76	90	60	70	NIL	68	97	65	76	NIL
14																														
15	87	90	59	69	2	87	90	57	68	2	89	93	60	71	2	91	87	54	65	2	92	86	53	64	1.8	89	87	54	65	1.8
16																														
17	58	90	57	68	1.4	50	90	58	69	1.4	78	90	60	70	1.6	82	89	61	70	1.6	79	90	58	69	1.4	81	89	58	68	NIL
18	61	95	59	71	1	68	96	58	71	1.2	65	94	58	70	1.2	67	92	58	69	1.2	63	91	56	68	1.2	63	88	55	66	1
19																														
20	79	133	84	100	NIL																									
21	71	95	62	73	NIL	72	91	60	70	NIL	75	98	66	77	NIL	78	100	60	73	NIL										
22																														
23	85	122	89	100	NIL																									
24																														
25	78	128	94	105	NIL																									

SL.NO	INTRAOP EVENTS						
	HYPER TENSION	HYPO TENSION	ARRYTHMIAS	ISCHEMIA	TACHYCARDIA	BRADYCARDIA	DURATION OF SURGERY
1	N	N	N	N	N	N	79
2	N	N	N	N	N	N	65
3	N	N	N	N	N	N	82
4	N	N	N	N	N	N	60
5	N	N	N	N	N	Y	75
6	N	N	N	N	N	Y	50
7	N	N	N	N	N	N	85
8	N	N	N	N	N	N	102
9	N	N	N	N	N	N	95
10	N	N	N	N	N	N	78
11	N	N	N	N	N	N	90
12	N	N	N	N	N	N	75
13	N	N	N	N	N	N	82
14	N	N	N	N	N	N	76
15	N	N	N	N	N	N	67
16	N	N	N	N	N	N	80
17	N	N	N	N	N	N	70
18	N	N	N	N	N	N	85
19	N	N	N	N	N	N	72
20	N	N	N	N	N	N	85
21	N	N	N	N	N	N	96
22	N	N	N	N	N	N	45
23	N	N	N	N	N	N	55
24	N	N	N	N	N	N	92
25	N	N	N	N	N	N	80

SL.NO			RAMSAY SEDATION SCALE			
	FROMME BOEZART SCALE	AWAKENING TIME	15	30	45	60
1	1	5	2	2	2	2
2	1	6	3	2	2	2
3	1	4	2	2	2	2
4	2	6	3	2	2	2
5	2	3	2	2	2	2
6	2	8	3	3	2	2
7	2	10	3	3	2	2
8	1	5	2	2	2	2
9	1	6	3	2	2	2
10	2	4	2	2	2	2
11	1	5	2	2	2	2
12	2	4	2	2	2	2
13	1	4	2	2	2	2
14	2	5	2	2	2	2
15	2	3	2	2	2	2
16	1	4	2	2	2	2
17	1	6	3	3	2	2
18	1	6	3	2	2	2
19	2	5	2	2	2	2
20	1	8	3	3	2	2
21	1	3	2	2	2	2
22	1	4	2	2	2	2
23	2	4	3	3	2	2
24	2	4	2	2	2	2
25	1	6	3	3	2	2

Abstract

Study objective: The aim of this study was to evaluate the effect dexmedetomidine infusion on the requirement of Isoflurane to produce controlled hypotension (mean arterial pressure of 60-70mmHg), quality of bloodless surgical field, duration of surgery and the awakening time in patients undergoing Functional Endoscopic Sinus Surgery (FESS).

Design: Prospective randomized placebo-controlled study.

Methods: 50 ASA I Patients age 18-60 years diagnosed having chronic sinusitis scheduled for FESS under general anesthesia were divided into two groups. Group D: Received 10-15 min prior to induction of anesthesia 1 μg/ kg iv bolus Dexmedetomidine followed by an infusion of 0.5 μg/ kg/ hr. Group C: Received 10-15 min before induction of anesthesia a normal saline rate similar to group D. Following a uniform premedication all patients were induced with inj .propofol and relaxed with inj. vecuronium bromide. After successful tracheal intubation, anesthesia was maintained with 66% nitrous oxide + 33% oxygen + isoflurane titrated to achieve a mean arterial pressure [MAP] of 60-70mmHg. Isoflurane and dexmedetomidine/saline infusion was stopped 10-15 minute prior to end of surgery. The residual neuromuscular blockade was reversed with neostigmine and glycopyrrolate. If at any stage heart rate was found to be less than 50 beats/ min, 0.3 mg

atropine was administered every 2 –3 min till it reached above 60 beats/ min. Concentration of isoflurane was recorded in percentage every 5 min in the intraoperative period. This was averaged for analysis. Intraoperative surgical field was assessed by using 6 point Fromme-Boezaart scale. Awakening time in min [clearly telling his/her name] from the end of tracheal extubation was also recorded. Student "t" test was used to analyze the data statistically. P<0.05 was considered significant.

Results: A statistically significant (p-<0.001) reduction in intraoperative isoflurane requirement in patients receiving dexmedetomitine infusion (0.387±0.102) in comparison to those receiving placebo (1.7±0.211). Both the group provided better visualization of the surgical field. The duration of surgery was statistically (p_ .004) low in Group D (76.84±14.174) compared to group C (94.1±25.083). The awakening time in min was statistically (p-0.001) low in group D (5.12±1.691) compared to group C(9.72±1.100).

Conclusion: Dexmedetomidine infusion helps in achieving a targeted reduction in MAP reduced intraoperative Isoflurane requirement, better blood less field, and faster awakening in patients undergoing Functional Endoscopic Sinus surgery.

Key words:

Dexmedetomidine , FESS, controlled hypotension.